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CARDIAC RUPTURE

REPORT OF 12 CASES FROM THE SOUTHERN BAPTIST HOSPITAL, NEW ORLEANS, DURING A 10 YEAR INTERVAL

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While cardiac rupture can be produced by a diversity of clinical conditions, such as trauma, and rarely by pyogenic abscess, tuberculous lesion, gumma, echinococcus cyst, and malignancy,[1] it is more commonly a complication of acute myocardial infarction. It has been reported by Freedman and White [2] to occur in 9.5 per cent of cases of recent myocardial infarction from a large general hospital. Other surveys by Oblath, Levinson, and Griffith,[3] and Edmundson and Hoxie,[4] report a comparable incidence of rupture, 7.11 and 8.6 per cent, respectively.

We thought it would be of interest to report the cases from a large private hospital in this area as, to our knowledge, no such report has been made previously.

Of special interest and importance to the practitioner are the conditions which predispose to cardiac rupture, and the micropathology of the healing process after infarction. These will be discussed, as we feel that such knowledge is important in the prevention of this fatal complication.

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FINDINGS

A ten year survey of the records of Southern Baptist Hospital (1944-1953, inclusive) reveals 1315 consecutive autopsies, among which 114 (8.7 per cent) were found to have acute myocardial infarction as the cause of death. Twelve cases of cardiac rupture were found (11 into the pericardium, and 1 interventricular), an incidence of 10.5 per cent of all acute infarcts autopsied.

Ages of these patients ranged from 54 to 85 years; an average age thereby of 68.7 years.

All patients were of the white race.

The sex ratio of this group of cases is worthy of further comment, being, contrary to expectations, predominantly female (8 female, 4 male). This will be elaborated upon.

Among the 12 patients in this report there were only 2 whose weight-height ratio could unequivocally be called normal. Five were of borderline overweight (one of whom was described clinically as "obese"), and 5 were definitely obese.

Past History: In several of the cases the severity and short duration of the final illness precluded any evaluation of previous health. Hypertension of undetermined duration was recorded in 4 of the cases, previous infarct in 1, angina in 3, "long-standing" coronary disease in 1, and slight cerebrovascular accident in 2.

In none of these cases was the diagnosis of cardiac rupture made premortem, although it was suspected in 2 cases at time of death.

Myocardial infarction was diagnosed

TABLE 1
CARDIAC RUPTURE
SOUTHERN BAPTIST HOSPITAL, NEW ORLEANS
1944-1953, inclusive

Case	Age	Sex	Obesity	Past History	ECG	Duration from onset to death	Blood Pressure	Type and location of pain	Activity at estimated time of rupture	Location of infarct	Size and location of rupture	Amount of hemopericardium	Size of heart	Degree of coronary sclerosis	Old Infarcts
1. A.S.	76	M	Slight	----	No	4 days	120/64	Severe epigastric with nausea	?	Posterior left lateral	Same (size not given)	Pericardium filled	---	Extreme	No (fibrosis)
2. B.N.	70	F	Moderate	High B. P. for years	Anterior infarction	24 hours	166/100	Viselike precordial; radiation to rt. arm	?	Occlusion of lt. ant. descending coronary	2 cm. slit along course lt. ant. desc.	300 cc.	"Not abnormal in size"	----	Yes
3 A.C.	71	M	No	Long standing coronary disease	No	19 hours	124/80	Precordial	?	Occlusion of lt. ant. descending coronary	4 x 1 cm. apex of lt. ventricle	Filled	---	Moderate	--
4. B.F.	69	F	Slight	High B. P. Left hemiplegia	No	Unknown: disoriented	195/130	(?) None (gasping for breath)	Very active. Died just after a meal	Apex	1 cm. Apex	100 cc.	440 grams	Moderate	No
5. E.A.	63	F	Definite	Transient left hemiplegia, 3 yrs. prior PI	Posterior infarction	12 days	240/120 110/80	Substernal and left arm	Talking, joking, developed pain and expired	Posterior apex (left ventricle)	Upper portion rt. vent. 1.5 cm. from lt. vent.	Filled	450 grams	Moderate	No
6. M.H.	85	F	Slight	----	No	14 days	120/84	Cramping abdominal, 1 day	Asked for bedpan	Occlusion of lt. ant. descending coronary	.25 x 2.5 cm. Apex	1000 cc.	450 grams	Moderate	No
7. M.C.	75	F	Slight	"Heart attack" 6 yrs. prior to PI	No	13 hours	120/90	Precordial radiation to both shoulders	Restless and vomiting entire time in hosp.	Left anterolateral extending to septum	1.5 cm. irreg. laceration lt. ant. lat.	200 cc.	310 grams	Extreme	No
8. F.D.	58	M	Definite	----	3 done: 1. Normal 2. Pericarditis 4. Effusion	5 days	170/100 100/70	Precordial radiation left shoulder, left scapula	Dyspnea	Postero-lateral	3 cm. Postero-lat. lt. vent.	Filled	475 grams	Slight	No
9. L.S.	68	F	Definite	Similar pain 7 yrs. prior PI (bed 3 days)	Developing anterior infarction	11 hours	134/92	Chest, radiation lt. arm; both hands numb	Dyspnea	Anterior apex	2 small defects, (2 mm.) just above apex ant.	200 cc.	400 grams	Slight	No
10. L.T.	54	M	Definite	----	Anteroseptal infarction	(?) Died 7 hrs. after admission	124/70	Chest pain (not fully described)	Sleeping 15 min. before death	Anterior apex	5 cm. Ant. left ventricle	Surrounded by blood	450 grams	Moderate	No
11. A.R.	65	F	Definite	----	Extensive anterior infarction	53 hours	140/80 110/70	Chest pain	Sudden chest pain then shock	Anterior left ventricle and septum	1 cm. through septum and into pericard.	Massive clot	275 grams	Minimal	No
12. C.S.	71	F	No	----	Extensive anterior infarction	11 hours	95/68	Severe generalized abd. pain more on rt. Nausea & vomiting	?	Occlusion of lt. ant. descending coronary	1.5 cm. Septal perforation near apex	30-40 cc. bloody fluid	300 grams	Extreme	No

clinically in 9 cases, of which 5 had electrocardiographic confirmation: 3 anterior, 1 anteroseptal, and 1 posterior infarction. One clinically unsuspected infarct was demonstrated by an ECG taken ten minutes prior to death (Case No. 12: tracing, which showed extensive anterior infarction, was developed and interpreted postmortem).

The two cases not diagnosed either clinically or electrocardiographically will be discussed in a little more detail:

Case No. 6 (white female, M. H.) presented symptoms of productive cough and wheezing, and was responding well to therapy for congestive heart failure until her sudden respiratory arrest after asking for a bedpan. Infarction and myocardial rupture were unsuspected prior to autopsy.

Case No. 8 (white male, F. D.) was a diagnostic problem for eight months, with pain in the right upper quadrant, right flank, back, and legs; admission diagnosis was "disc syndrome". Five days prior to death he developed precordial pain radiating to the left shoulder. The diagnosis of renal tumor with venous spread to the heart was considered. Myocardial infarction and rupture of the ventricle were not diagnosed until autopsy. (Incidentally, the diagnosis of renal tumor with invasion of the veins was confirmed, but cardiac involvement by tumor was not found.)

The duration of illness in these cases, from onset of symptoms compatible with myocardial infarction until death, ranged from seven hours to fourteen days; 8 ruptured within the first four days. In one, symptoms were not present, and death was apparently instantaneous.

Elevation of blood pressure during the terminal illness was present in only 4 of the 12 cases; in 7 the blood pressure was normal or borderline, and in only 1 was hypotension found. These were, however, isolated readings taken at variable times during the final illness, and are not necessarily representative of the patients' usual blood pressure range.

In only 1 case was there no record of pain; that patient, however, was disoriented and hemiplegic, and a satisfactory history was not obtained. Of the remaining 11, character of the pain can be classified as follows:

Precordial: 4
- 1 with radiation to right arm
- 1 with radiation to left shoulder
- 1 with radiation to both shoulders
- 1 with no radiation

Substernal: 1, with radiation to left arm

"Chest": 3
- 1 with radiation to left arm; hands numb
- 2 with no radiation

Epigastric: 1, with severe nausea

Abdominal: 2
- 1 generalized, cramping
- 1 generalized, more on right; nausea and vomiting

It is interesting to note that only 2 of the 11 had nausea, and only one vomited; that patient (No. 12) was suspected to have either gastroenteritis or biliary tract disease, and expired undiagnosed.

Activity at the estimated time of rupture is difficult to ascertain. All cases were theoretically at bed rest; however, the mere physical limitation to bed does not mean that the patient is actually at rest. Several of these patients had been under physical or mental "stress" just prior to rupture. In 3 cases there is no record of activity, 2 were apparently quiet (one "sleeping" fifteen minutes before being found dead, and one stuporous but dyspneic). The remaining 7 patients had had variable and difficult to evaluate stress, such as chest or abdominal pain, urge to use the bedpan, talking and joking, and actively consuming a meal.

At autopsy all cases were found to have acute myocardial infarcts with rupture through the area of involved muscle. The anatomic locations of the infarcts are shown in Table 2.

Rupture occurred through the infarcted area in all cases. The case of interventricular rupture (No. 12) had ECG evidence of extensive anterior myocardial infarction. Upon gross examination the

TABLE 2
ANATOMIC LOCATION OF INFARCTS

Apical	1
Posterior apical	1
Anterior apical	2
Anterior left ventricle	5
Anterolateral left ventricle	1
Posterolateral left ventricle	2

infarct was found to involve the apical portion of the right ventricle, distal septum, and anterolateral portion of the left ventricle. A 1.5 cm. perforation had occurred in the distal septum, and blood had extravasated into the wall of the right ventricle; rupture of this area was impending.

The other 11 cases had definite rupture of the myocardium, with communication between the intracardiac and the extracardiac (pericardial) spaces. The defects ranged in size from 2 mm. to 4 x 1 cm. Case No. 9 had two small defects, each approximately 2 mm. in diameter, penetrating through the wall of the heart. None were described as "blowout".

The volume of blood in the pericardium in the 11 cases of frank rupture was measured in 5 cases, and ranged from 100 cc. to 1000 cc.; in the remaining 6 cases the pericardial cavity was reported to be "filled with blood".

Heart size and weight were not recorded in 2 cases; in a third case it was stated to be "not abnormal". The 9 cases weighed ranged from 275 to 475 grams. If normal heart weight is considered to be 250 to 350 grams, 6 of this group were unequivocally enlarged; the 3 within the above "normal limits" (275, 300, and 310 grams), when evaluated according to the patients' body size (height and weight) were also enlarged.

The degree of coronary sclerosis was described as minimal or slight in 3, moderate in 5, and severe or extreme in 3. One was not graded by the pathologist.

Autopsy evidence of previous or "old" infarction was present in only 1 case; a second case was reported as showing much fibrosis, but an actual area of infarction was not found. The remaining 10 cases had no evidence of former occlusion or infarction, although 1 of these gave clinical history of a "heart attack" six years earlier.

COMMENT

The age range (54 to 85 years) and the average age (68.7 years) of our cases compares favorably with other series reported in the literature.[2, 3, 5-9] Spontaneous rupture following acute myocardial infarction rarely occurs under 50 years of age.[3, 6] As reported by Kelley et al[10] this disaster has occurred as early as 3 months of age. However, the underlying pathology in their case was an anomalous left coronary artery that arose from the pulmonary artery.

It is worthy of comment that in our series there was a predominance of female patients. Oblath et al[3] and Gans et al[8] also reported groups of cases in which there was a predominance of women. In large collections of cases from the literature such as that reported by Krumbhaar and Crowell[6] there is a predominance of males. When compared to the incidence of acute myocardial infarctions there is, however, a higher relative incidence of spontaneous heart rupture in the female.[3, 8, 11] Wessler et al[11] attributed this to the lower incidence of coronary disease and the higher incidence of hypertension among women.

Underlying and contributory causes for spontaneous heart rupture have been the subject of much discussion. This catastrophe is rare except in the presence of pre-existing acute myocardial infarction.[2, 3, 5, 9, 11] Several authors[3, 4, 6-9, 11] have studied and discussed the reasons why some hearts with acute infarction rupture, and others do not rupture.

It is now well recognized that increased activity following acute myocardial infarction definitely increases the incidence of heart rupture. Jetter and White[9] in their report of cases occurring in mental institutions showed a much higher incidence of heart rupture (73 per cent of deaths from acute myocardial infarction) among these people in whom activity was totally uncontrolled because of their mental illnesses. Others have commented upon the

increased incidence of heart rupture with increased activity following acute infarctions.[3, 5, 7, 11] It is interesting to speculate upon the effect of the "armchair" treatment of myocardial infarction as advocated by Levine and his group[12] upon the incidence of heart rupture in these individuals. Strict adherence to their routine probably would not influence the rate of occurrence of heart rupture because, as they emphasize, they do not advocate early ambulation, but rather a different approach to management at rest. The present trend toward early ambulation of the patient who has the so-called "small infarct" and does well clinically may prove to be a dangerous procedure when more follow-up studies have been done. Edmondson and Hoxie[4] pointed out that in their series the infarcts of ruptured hearts tended to be smaller and more necrotic than those that did not rupture. The infarct was frequently surrounded by otherwise healthy muscle. These authors also indicate that in the presence of the scar of a healed infarct the incidence of heart rupture is only one-fourth as great as would be expected.

None of our patients had been ambulatory between onset of the infarction and death. One patient died shortly after eating, one while talking to a relative, one shortly after asking for a bedpan, and a fourth had been vomiting and restless during the entire hospital stay. All others were presumably at rest, and one patient was asleep at the time of death. Hence, activity does not appear to have been a prominent causative factor in our cases.

Sustained hypertension following infarction has been shown by several authors[3, 4, 5, 7, 8, 11] to increase the probability of heart rupture. Howell and Turnbull[7] and Edmondson and Hoxie[4] compared blood pressures in patients dying of cardiac rupture and patients with acute myocardial infarction who died without rupture. In both instances the authors were able to demonstrate a higher percentage of cases with blood pressure above 140/90 among those who died of heart rupture. Oblath et al[3] state that with persistent hypertension the danger of heart rupture complicating myocardial infarction is increased threefold. In 4 of our cases (33.3 per cent) blood pressures above 140/90 were recorded in the hospital record. In a fifth case a diastolic pressure of 92 was recorded.

Eight (66.7 per cent) of our cases ruptured within four days (seven hours to four days) of the onset of the infarct, one on the fifth day, one on the twelfth day and one on the fourteenth day. In the remaining case it was impossible to tell from the record when the infarct occurred, because the patient was disoriented from the time of admission and no history of onset of infarct was available. Oblath et al[3] found that 45 per cent of their series ruptured within the first three days and 78.75 per cent ruptured by the twelfth day. Howell and Turnbull[7] reported 8 cases, all of which ruptured within eleven days. Friedman and White[4] reported 10 cases, all of which ruptured within two weeks. Diaz-Rivera and Miller[5] found that their 5 cases died within thirteen days of onset of infarction. Seventy-eight per cent of those in the series reported by Edmondson and Hoxie[4] died between the third and twelfth days. The time of rupture in our group follows essentially the same pattern as reported by these authors.

When one reflects upon the microscopic pathology of acute myocardial infarction as described by Mallory et al,[13] it is expected that the greater percentage of heart ruptures should occur within the first few days after onset. We feel that it is appropriate to review some of their findings at this point. Necrosis begins immediately, is greatest at two to six days, and may last as long as four months. Hemorrhage is variable and usually limited. Polymorphonuclear cells appear immediately, increase to the fourth day, then gradually decrease to disappear about the fourteenth day. Ingrowth of capillaries and connective tissue cells begins about the fourth day. Fibrin is absent. Removal of necrotic tissue begins about the fourth day. Collagen first appears about the

twelfth day and reaches a maximum by two to three months. Collagen especially, and connective tissue, add strength to the healing infarct. However, they point out that the rate of healing of an individual infarct is influenced by its size, location, and the adequacy of collateral circulation. From these findings one readily sees that the infarcted area is weakest during the first few days. If polymorphonuclear cells increase liquefaction, then the fourth to the sixth days should be the time at which the infarct is weakest and hence the time at which most heart ruptures occur. The times of rupture of our group fall into a pattern that is compatible with this pathological description.

The pain experienced by patients in this group was no different from the usual severe pain of coronary occlusion. Two of our patients experienced severe pain at the time of death. No diagnostic symptoms were revealed in the histories of these patients. Bishop and Logue[14] reported a case of external rupture that developed a systolic murmur and thrill suggestive of septal rupture. Askey and Cherry[15] in their study attempting to evaluate diagnostic features felt that an antemortem diagnosis of external rupture is very difficult but that septal rupture may be diagnosed prior to death. It is therefore felt that the signs and symptoms of heart rupture are indistinguishable from those of acute coronary occlusion, and that heart rupture is a difficult antemortem diagnosis.

The effect of digitalis therapy upon heart rupture in myocardial infarctions is a much discussed subject. Oblath et al[3] felt that digitalis had no effect in their series. Two of our patients were taking digitalis preparations at the time of death. It is our feeling that when there is a definite therapeutic need for digitalis in the presence of an acute infarction that it should be used.

Our group of cases gives no help in determining the possible effect of anticoagulants upon heart rupture. Only one of our cases received an anticoagulant (ethyl biscoumacetate), and only a single dose (150 mgm.) approximately three hours before death. This drug could have had little or no effect in that period of time. Anticoagulant therapy in acute myocardial infarction has been widespread in recent years.[16] Russek and Zohman[17] in a recent survey found that there is still much conflicting opinion about the use of these drugs. As reported by Goldstein and Wolff[18] hemopericardium may develop as a complication of anticoagulant therapy. Flaxman[19] reports that these drugs lead the list for fatalities caused by oral medications. These fatalities are, as one would expect, almost all from hemorrhagic complications. Gans[8] mentions the possibility that anticoagulants may influence the incidence of heart rupture. Clagett et al[20] feel that anticoagulants do not increase the rate of rupture, but suggest that analysis of a larger series of cases is needed before drawing final conclusions. Waldron et al,[21] in a recently reported study, were able to demonstrate a significantly higher incidence of heart rupture following acute infarction in cases that were treated with anticoagulants. They also demonstrated a higher incidence of hemopericardium without rupture when anticoagulant therapy was used. Anticoagulants are valuable drugs in the treatment of acute myocardial infarction, but one must be aware of their dangers before prescribing them.

The rupture in our cases were linear tears for the most part. Ten were of this type, one was septal and one showed two very small (2 mm.) perforations. The size, shapes, and positions of the ruptures found in our cases are similar in most respects to those reported by others.[2-9] No instances of the so-called "blow-out" rupture and no instances of right ventricular rupture were seen in our group.

SUMMARY AND CONCLUSION

1. Twelve cases of cardiac rupture from the Southern Baptist Hospital, New Orleans, are reported and described. The incidence of rupture was 10.5 per cent of acute infarctions.

2. It would seem that since most ruptures occur within the first week and

nearly all within the first two weeks, and since the incidence of rupture is increased by unrestrained activity, the physician is justified in insisting on at least three weeks of complete rest. This would apply equally to the so-called small infarct, particularly when the blood pressure remains high after infarction.

3. Anticoagulants carry a definite risk in addition to the systemic effects of the drug. The occurrence of cardiac tamponade, as well as the increased frequency of rupture, limits the usefulness of this drug in the care of the patient with acute myocardial infarction.

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RADIATION TREATMENT OF NONMALIGNANT CONDITIONS *

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The use of roentgen rays and radium in the treatment of malignant conditions has assumed such a large place in the therapy of these lesions that many forget that there are nonmalignant conditions which can be equally well treated with these modalities. The dermatologist, of course, is well acquainted with the value of roentgen rays in the treatment of many nonmalignant skin conditions, but many physicians are not sufficiently acquainted with the use of roentgen rays for these nonmalignant disorders to gain for their patients the benefits that will accrue from careful treatment. In the time at our disposal today I hope to go over with you a few of the conditions which can definitely be benefited by radiations, and which do not come under the heading of malignant tumors. The first group which I wish to discuss can be classified as tumors, however. These may be of the pituitary gland, of the nasopharynx, of bones, such as giant cell tumors, hemangiomas, lymphangiomas, keloids, etc.

PITUITARY ADENOMA

Pituitary adenomas are divided into three main groups: The acidophilic, basophilic and the chromophobic. The acidophilic pituitary adenoma produces the growth hormone. If this occurs before the epiphyseal lines are closed the patient becomes a giant. If he has already reached his growth and the epiphyses are closed, he becomes an acromegalic. Cushing's basophilism, so-called, is apparently pro-

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duced by basophilic adenomas of pituitary. These patients have the cardinal symptoms of hirsutism, abdominal striae, peculiar fat distribution, hypertension, etc. The chromophobic adenoma is nonhormone producing, but by its size may cause sufficient pressure on the anterior portion of the pituitary to decrease its normal hormone production. By their size, both acidophilic adenomas and chromophobic adenomas may cause pressure symptoms on the optic chiasm with bitemporal field cuts. This is almost always true with the chromophobic type, but occurs in only about 50 per cent of the acidophilic tumors. The chromophobic adenoma produces increase in the size of the sella in almost all cases, whereas the acidophilic produces it in only about one-half, while the basophilic tumor never shows an increase in the size of the sella turcica. In my opinion, the best primary treatment for pituitary adenomas is radiation. It should be delivered through several fields about the skull, given slowly until an estimated tumor dose of from 2500 to 3000 roentgens has been delivered. It is of value to have visual field studies taken at weekly intervals during treatment in order to ascertain whether or not there is gross change. It should be remembered, however, that an increase in the field cuts during treatment is of no significance and should not make one stop the treatment. It is of the utmost importance, however, to have the patient return in about two months for further studies of the visual fields and observation of his general condition. If, at that time, there is increase in the visual cuts, the patient should be immediately referred to the neurosurgeon for his care. I think that all of *these* patients should be operated upon, since the tumor which has been treated is likely to be cystic, and these cystic tumors do not respond well to radiation. If, however, the visual field cuts have not increased or have decreased, then the patient may be watched. Approximately 70 per cent of patients with these tumors will do well with radiation, and ordinarily a good result remains permanent. We have, however, had two or three cases in which there was a good result for from one year to five years, with then a recurrence of the tumor as shown by increasing symptoms. These patients should then be operated upon. In no case have we observed any hypopituitarism from irradiation.

CRANIOPHARYNGIOMA

Craniopharyngioma or suprasellar cyst is a lesion which is not ordinarily considered radiosensitive. It occurs in childhood or early adult life and is a developmental defect. Its symptoms are those of a mass in or above the sella. It is usually at least partially calcified. There is reason to believe that if the fluid can be aspirated and irradiation given to prevent the re-formation of fluid, this may be successful. It has proved to be so in some cases with the use of roentgen rays, and a recent personal communication from Woods and Hiebert indicates that injection of radioactive gold into the cyst may well prevent the re-formation of fluid by the radiation from the Au-198. This may be a very valuable addition to our methods of treatment of this condition.

NASOPHARYNGEAL FIBROMA

Nasopharyngeal fibroma or sclerosing capillary hemangioma of the nasopharynx is a lesion which occurs in young males from 12 to 18 years of age. These tumors may be firm or somewhat friable, may bleed without trauma, and are found to be rather vascular at the time of biopsy. They destroy adjacent tissue only by pressure and are not malignant tumors. They are said by many to regress spontaneously with the advent of puberty, but in our experience, this is not usual. Even the cases to whom we have given testosterone and who have had early maturity because of this have shown no regression of their tumors. The basic treatment of these lesions is surgery, but this can usually not be done until the lesion has been so sclerosed and reduced in size by radiation that operation becomes feasible. The treatment is best given by interstitial radium needles or by radon seeds, with a delivered dose of 4000 to 5000 gamma

roentgens. Surgery may then be done without danger of exsanguinating hemorrhage.

CAVERNOUS HEMANGIOMAS

Cavernous hemangiomas are not an uncommon lesion in infancy and young childhood. In our experience they occur at least 85 per cent in girls. They may be present at birth or appear shortly after. They may show rapid, spontaneous disappearance or they may continue to grow rapidly. It is said by many that these all will disappear spontaneously without any treatment. Although I am sure that this is frequently the case, there are many whose lesions increase to such size that something must be done for them. I think any hemangioma which is obviously increasing in size should be given some type of treatment, preferably radiation. Our treatment generally consists of giving about 300 roentgens, using a Chaoul contact therapy unit, although conventional low voltage units may be used. We then wait six to eight weeks before seeing the patient again. If there is any sign of regression of the lesion, no further radiation is given. If there is still an advancing edge, the treatment will be given to this active portion of the lesion. All that we try to do is to have the lesion start in the proper direction and then let the child take care of it naturally. A minimum amount of radiation is desirable. Some large and deep hemangiomas which are fed by large vessels will not respond satisfactorily to superficial treatment. The smaller vessels may become sclerosed but the larger ones will not, in general, be touched, and many of these have to be taken care of by surgical excision. In regard to lesions around the eye or in rather inaccessible places where roentgen therapy is difficult, irradiation may be given by the implantation of radon seeds. Gold filtered radon seeds with a content of about one-tenth to fifteen (0.1 to .15 mc.) hundredth of a millicurie are usually sufficient to cause rapid regression of the lesion.

GIANT CELL TUMORS OF BONE

Giant cell tumors of the bone have for many years been treated by irradiation. Formerly, they were treated with large doses and some ill effects have resulted, even to the development of osteogenic sarcoma in the irradiated site. Giant cell tumors may also be subject to malignant change. This was either present at the time of the first examination and remained unrecognized, or it developed after a course of treatment, either surgical or radiologic. My present concept regarding the best treatment for accessible giant cell tumors is that they should be treated by surgery, and irradiation used only if surgery proves to be ineffective. For lesions in relatively inaccessible portions of the body, such as the spine or portions of the pelvis, it seems that radiation is preferable as the primary treatment. In those cases we give from 600 to 1000 roentgens as measured in air, giving 200 roentgens a day on four or five consecutive days. Further treatment is not given for at least two months, and then only if there is no good evidence of healing of the lesions.

KELOIDS

Keloids are not considered generally as basically tumors, but they are masses of fibroblastic tissue which develop at the site of trauma and which tend to recur after excision. If these can be seen in the early succulent stages, irradiation is very valuable. A single dose of about 800 air roentgens given to the keloid itself with protection of the surrounding skin is quite effective. If the lesions are large, such as follow extensive burns, the dose is reduced to about 600 roentgens. Even in old sclerosing cases, which have developed intractable itching, a small amount of radiation—300 to 400 roentgens—may be sufficient to relieve the patient of all his symptoms. When a keloid is excised and replaced by a linear incision, it is wise to irradiate the incision with *about* 800 roentgens to prevent further keloid formation.

BURSITIS

A great deal has been written regarding radiation treatment of bursitis. I think frequently, however, that this term is a

misnomer and should be called "painful shoulder or elbow," because many times there is no bursitis but pain is or may be due to degenerative changes in the supraspinatous tendon. Regardless of the exact etiology, this lesion is a painful and disabling one, and for days may prevent normal rest at night and function during the day. There are those skeptics who say that no matter what one does for this condition a good result follows, or vice versa, and I am sure that many times this is the case if one waits long enough. For one who is in severe pain and disability, however, the application of a small amount of radiation—100 roentgens or possibly 200 roentgens, or even only 50 roentgens if the process is very acute—gives almost a miraculous relief of pain, and usually the pain does not recur except as a new episode. Orthopedists are somewhat loathe to use this method of treatment and prefer heat, massage, and both passive and active motion. Most of the cases which we receive from them have gone into the subacute or chronic stage when radiation therapy is reduced in its effectiveness to about 50 per cent. If one is to use radiation, however, it is important to keep the shoulder not immobilized but confined to the range of motion which is not painful to the patient.

SPONDYLITIS

Another condition of the bone and joint system is ankylosing spondylitis of the rheumatoid type — so-called Strumpell-Marie or Von Becterew. This condition, as you know, is the one which produces a great deal of pain and disability in the spine and which eventually leads to a "poker" spine. Irradiation in the early cases of this disease are frequently and almost uniformly followed by prompt relief of pain which relief may last for a matter of years. Instead of having excruciating pain, spasm and disability, the patient may have only a mild ache or stiffness. I am convinced, also, that radiation treatment for this disease not only affects the pain, but changes the course of the process. The treatment consists of three, four, or five times 200 roentgens, as measured in air, given to the affected area. These treatments may be given on consecutive days or once a week. The benefit is indeed striking at times. The pain from involvement of rheumatoid arthritis of the extremities can also be alleviated by small doses of radiation, but the duration of the relief seems not to be nearly as long as with the spondylitis. The disabling pain from osteoarthritis or degenerative joint disease is also sometimes relieved, but this is only in a small fraction of the cases. I would suggest, therefore, that in all cases of spondylitis in the young individual, a course of radiation be given with the hope that not only the pain may be relieved but the course of the disease altered favorably.

SYRINGOMYELIA

Syringomyelia is a condition which consists of a diffuse gliosis of the spinal cord with resultant cyst formation and the inevitable symptom complex which accompanies this condition. The only method of surgical attack is the opening of large cavities and evacuation of their fluid content. This, of course, is a temporizing measure since the cysts will again fill with fluid and cause recurring pressure. It has been found in some of these cases that irradiation given in the amounts of six times 200 air roentgens to the involved portion of the cord will relieve the symptomatology and hold it in abeyance for a matter of many months or even years. When there is a recurrence of symptoms the treatment may be repeated. It is well to warn, however, that not more than about three such courses of treatment should be given and then only with the greatest caution, since they may be followed by a cutaneous breakdown over the spine. I have, however, seen patients come up to our department bedridden and by the time they were finished with their treatment, were walking back and forth. This rapid improvement, however, is unusual but many cases of lesser degrees of improvement have been encountered. It is well worth the trial.

UTERINE BLEEDING

The question of uterine bleeding is suf-

ficiently broad so that it might cover several hours of dissertation. I shall, therefore, discuss only briefly some of the indications for the use of radiation in the treatment of uterine bleeding of nonmalignant character. As a general statement, I think it is fair to say that only climacteric bleeding or bleeding associated with fibroids at the approximate age of menopause can justifiably be treated by irradiation. At the outset, however, I would like to warn against the indiscriminate use of irradiation for uterine bleeding until every vestige of a chance of the bleeding being due to a malignant tumor has been eliminated. Careful pelvic examination and careful segmental curetting of the uterine cavity and the endocervix should be done to rule out a malignant tumor. Omission of this simple examination may lead to disastrous results. There is considerable discussion these days regarding the value of roentgen sterilization. The main consideration being raised is that perhaps this type of treatment does not eradicate all ovarian function. This may perhaps be true, but since we are talking about uterine bleeding and not castration, I can say with assurance that a dosage to the ovaries of around 600 to 800 roentgens is sufficient to stop menorrhagia or menometrorrhagia. I believe that roentgen sterilization is superior to sterilization by intracavitary radium, particularly if there are any uterine fibroids. These may so displace an ovary that the radiation from the uterus is ineffective and sterilization is not obtained. Also, the use of radium ordinarily requires dilatation of the cervical canal and I have seen cases where this procedure has resulted in a parametritis. Neither of these poor results follows external radiation properly given. I shall not discuss the use of roentgen rays in the treatment of the ovaries as an adjunct to the treatment of a malignant neoplasm. Under certain circumstances it may be completely justified to irradiate the ovaries in cases of endometriosis, however.

INFLAMMATORY CONDITIONS

For a great many years roentgen rays have been used in the treatment of certain inflammatory conditions. Before the discovery of the sulfonamides and the antibiotics, it was almost routine to treat such infections as erysipelas, boils, and certain carbuncles with small doses of irradiation. The results were quite striking. Before the advent of this chemotherapeutic age, also, many cases of pneumonia were satisfactorily treated by radiation. At present, however, it would be folly to take a patient from his bed in a ward to a department of Radiology in order to give him radiation therapy when he could be treated adequately and just as well, and if not perhaps better, in his own bed. There are, however, a few remaining inflammatory lesions which still seem to respond better to radiation than to any chemotherapy which is at present available. Among these I would like to mention only two. The first is acute or subacute thyroiditis. This condition, as you know, is manifest by swelling and pain in the thyroid gland accompanied by fever, difficulty in swallowing and general malaise. The BMR may be elevated and the patient present all the signs of thyrotoxicosis. An important diagnostic test is the radioactive iodine uptake studies which are exceedingly low in this condition. Antibiotics and other chemotherapeutic measures seem to have little, if any effect, on this condition. It responds promptly and well in almost all cases, however, to small doses of irradiation given to the affected gland. In the very acute cases we give 50 roentgens to each side of the thyroid to include the isthmus, using intermediate voltage. If, on the following day, there is evidence of improvement (and there usually is), the dosage is raised to 100 roentgens to each lobe of the thyroid. This is continued until a total of about 450 roentgens, as measured in air, are given to each side of the neck. The results in most cases are dramatic, although there are cases who have only partial subsidence of their symptoms. The febrile reaction disappears, the tenderness of the gland vanishes, but its size does not perceptibly change during the

course of treatment. Some of these patients may have recurring episodes in the opposite lobe if at the first time only one lobe of the gland is involved. A second course of treatment is well tolerated. Thyroiditis is frequently followed by hypothyroidism. When this occurs, it is well to remember that it is due to the disease and not the treatment.

The second inflammatory condition which I wish to discuss briefly is that of postoperative parotitis. This condition, as you know, is most commonly found in the elderly and debilitated patients following major operations, although it may occur in the young. There is gross swelling and tenderness of the parotid, either unilateral or bilateral, and the patient has a febrile reaction. The treatment of this condition is not confined to radiation, but radiation has a large place in its care. It is important that the patient have a proper fluid balance, that the parotid gland and duct be gently massaged toward the ductal orifice and that the patient be encouraged to chew and to suck acid foods, such as lemons, etc. These are very important parts in the treatment of this serious disorder. Results are not uniformly good, but this is usually because the patient is debilitated, elderly and in poor condition generally.

EYE LESIONS

For many years, low voltage roentgen rays and beta rays from radium have been used in the treatment of lesions about the eye. With the discovery of the radioactivity of Sr. 90 with its emission of beta rays, this has become the mainstay in the treatment of nonmalignant conditions about the eye. I shall discuss none of these in detail but merely mention that lesions as certain corneal ulcers, pterygia, new vascularization, small hemangiomas and lymphangiomas, etc., may be greatly benefited by judicious use of this form of radiation.

As you can see, therefore, from these few examples discussed briefly, radiation is an effective agent in the treatment of many nonmalignant conditions and deserves greater consideration in their care.

THE DIAGNOSIS AND MANAGEMENT OF CONGENITAL CYSTIC DISEASE OF THE LUNG

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Pulmonary cysts arising on a congenital basis have only recently become recognized as not only an interesting pathological entity, but as a fairly frequent cause of pulmonary disease and disability. Congenital cysts of the lung, more often than not, tend to masquerade as other pathological conditions. Many cases of unresolved pneumonia, refractory empyema, tuberculosis, suspected carcinoma, bronchiectasis, etc., are in reality infected congenital pulmonary cysts. Failure to recognize or consider this possibility often results in prolonged and ineffectual treatment.

In the American literature there was no interest in congenital cystic disease of the lung until Koontz's article appeared in 1925. However, Sauerbruch earlier wrote extensively of this entity in the German literature, and reported his findings in 58 resections performed in children. With increased recognition of this condition, much confused and conflicting thinking has resulted. There have been innumerable published classifications, with little attempt at clinical-pathological correlation. As a result, congenital cysts are often grouped under the acquired cysts of the lung. The latter should in reality be considered separately. Probably one of the best working classifications is that of Dickson:

I. True developmental or so-called congenital cysts of the lung.

II. Acquired cysts, or cystlike cavities of the lung.

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A. Cystic bronchiectasis
B. Other pulmonary cysts
   1. Pneumatoceles
   2. Abscess cysts
   3. Emphysematous blebs
   4. Emphysematous bullae

EMBRYOLOGY

When the developing lung buds grow out from the primitive foregut of the embryo, their normal development may be altered at any point. If this occurs, at an early stage, a fragment may be pinched off near the trachea, and a mediastinal bronchial cyst left. If a more substantial part goes astray, an entire sequestrated lobe may result. If alteration of normal development occurs peripherally, solitary or multiple smaller cysts are formed. The cysts are always lined by columnar, ciliated, mucus producing epithelium, and may or may not be connected to the bronchial tree. The cysts generally contain a turbid, milky fluid at birth; if there is a communicating bronchus, they will contain air. The communicating cyst is always a receptive focus for secondary infection.

There are associated congenital anomalies in about 15 per cent of cases, the most common being accessory lobe, aberrant systemic arteries, and anomalous venous drainage.

SYMPTOMS

The symptom complex of congenital cystic disease of the lung is best expressed by the natural history of the cysts themselves and is largely the symptoms of their complications.

1. Inflation
2. Infection
3. Bleeding

If neither of the three above sequelae occur, the patient may be symptom free for many years, but with the ever present possibility of later grave complication.

Inflation: The communicating bronchus may run a tortuous course through the cyst wall, and owing to some mechanical change, such as kinking or retention of mucus, it comes to act as a check-valve, allowing air to enter but not leave the cyst. This progressive inflation causes a "giant cyst". The pressure inside is positive, it may displace the mediastinum and by pressing on adjacent vital structures threaten the patient's life. They are most often seen in young children and in the newborn, who are easily embarrassed by the resultant compression of lung tissue. A giant cyst may be easily mistaken for pneumothorax, and needling the cyst may produce a resultant true tension pneumothorax. Recurring bouts of dyspnea and cyanosis in young children should make one aware of the possibility of underlying cystic disease.

Infection is probably the most common disabling feature and causes the entire gamut of symptoms that characterize pulmonary infection and are common to the infectious pneumonias, bronchial obstructive pathology, lung abscess, etc. If the bronchus communicates there will be expectoration of purulent secretion. There are always varying degrees of pleural pain, temperature elevation, cough, and generalized weakness. Unless the prime cause, cystic disease, is thought of and treated, the patient is often subjected to prolonged, expensive, and ultimately, ineffectual programs of therapy.

Bleeding is more often the result of low grade, chronic infection within the cavity of the cyst, and may assume threatening proportion. It is not unusual to see exsanguinating hemorrhage from these cysts, which show on dissection extensive arterial and venous collaterals within their walls, with marked increase in the calibre of these submucosal vessels. Bronchoscopy is most helpful in localizing the bleeding site, and should always be done, if possible, during the bleeding phase. Bleeding, so localized, may be the first clue to further study, in a patient who appears to have a normal chest film. Alarming hemorrhage may be the first symptom in a patient who has had benign appearing cystic disease by x-ray for years. I have recently resected a lower lobe in a patient who entered the hospital in shock from hemorrhage, at the age of 65, and who had had noted cysts within his right lower lobe for twenty years, with absolutely no antecedent symptoms.

The infected cyst may on occasions rupture into the pleural cavity, with resultant empyema or tension pneumothorax. A large cyst that has filled with purulent exudate is often indistinguishable from an empyema, and may give the identical symptoms.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis depends to a great extent on the x-ray appearances. If a cyst contains air, is solitary and large, it must be differentiated from pneumothorax, large emphysematous bullae, and from diaphragmatic hernia.

The smaller solitary cyst may resemble a thin walled tuberculous cavity. If the cyst contains fluid, or fluid and air, it may be mistaken for empyema, lung abscess, hydatid cyst, or solid tumor.

The multiple fluid filled cysts are commonly mistaken for saccular bronchiectasis, and the following table is helpful in their general differentiation. (Table 1)

The following diagnostic measures have been the most helpful to the thoracic surgeon:

A. Painstaking x-ray study:

Oblique views, lordotics, and stereoscopic technique will often disclose a rounded density or rarefaction when the standard PA view is read to be normal. Laminography is most helpful in delineating smaller irregular shadows, and will often reveal an air-fluid level where not suspected. Bronchography is a valuable adjunct, particularly in mapping the extent of cystic disease where there is bronchial communication. If the cyst is non-communicating, it will often show adjacent crowding of bronchi, and thereby outline the extent of the pathologic process.

B. Bronchoscopy:

Bronchoscopy is both a valuable diagnostic and therapeutic tool. Where there is bleeding or purulent expectoration, endoscopic examination will often localize the segment or lobe involved before the cysts have been discovered by x-ray. At the same time, it is helpful in ruling out other endobronchial pathology, particularly carcinoma. Prior to surgery or bronchographic examination, it is used to institute adequate bronchial lavage, and is particularly helpful in managing the patient who because of extent of disease or other reasons must be denied surgery.

C. Pulmonary function studies:

Lung volume studies are helpful in the instances of large inflated cysts, when one wishes to know if there is a bronchial communication.

TREATMENT

The ideal treatment of congenital cysts, whether they be the ballooning air cysts of infancy or the infected, bleeding cavities of later life, is excision with careful closure of the bronchial defect. In the larger mediastinal forms, it is occasionally possible to remove the cyst without sacrifice of lung tissue. Localized single or grouped cysts may be removed by segmental resection or lobectomy. Pneumonectomy for diffuse, unilateral involvement is occasionally necessary.

TABLE 1

	CYSTIC BRONCHIECTASIS	CONGENITAL CYSTIC DISEASE
Size:	Usually small. 2 - 4 cms. in diameter	Usually 4 - 5 cms. and much larger
Location:	Usually basilar lobes	Anywhere. Even paratracheal or mediastinal
State of Comm. Bronchus	Always show dilatation and thickening	Usually normal
Ass'd Parenchyma	Ass'd pneumonitis with or without atelectasis	Usually normal
Microscopic	Irregular roughened lining. Squamous metaplasia. Lining epithelium may be lost.	Stratified columnar. Ciliated, mucus producing

In all instances, the preservation of the maximal amount of functioning lung tissue must be the surgeon's aim. Bilateral disease need not necessarily deny the patient surgical treatment, but there are some instances in which the disease is so diffuse as to preclude consideration of excision.

The nonoperable cases are treated in the same manner as cases of diffuse, bilateral, saccular bronchiectasis. Though the results are palliative at best, these patients can be improved and their lives prolonged by judicious use of postural drainage, systemic and aerosol antibiotics, and the newer bronchial dilators and wetting agents. Bronchoscopy and lavage can offer striking relief to this palliative group.

CASE REPORTS

Case No. 1.— (See Figures 1 and 2) Patient is a 32 year old, married male, whose illness dated back to 1951, when he first began to complain of hemoptysis. X-ray at that time disclosed 2 cysts of the left lower lobe, one of which contained a fluid level. Bronchography failed to fill these cysts, and did not disclose other areas of cystic disease. No specific treatment was advised at the time. The patient continued to suffer intermittent progressive hemoptysis, malaise, left chest pain. Serial x-rays showed progressive enlargement of the cysts. The remaining examination was otherwise negative. During August 1953, patient suddenly became febrile, his cough increased, and he began to raise up to 3 oz. daily of bloody sputum. An x-ray now showed a round dense shadow at the left base, interpreted to be a filled cyst. He was given a course of antibiotics, postural drainage, and was bronchoscoped, the latter revealing blood clots coming from basilar orifices of the left lower lobe. At operation, in September 1953, he was found to have a dense, firm left lower lobe, with considerable pleural thickening. The lobe contained two large cysts, one of which was filled with old, changed blood clots. The upper lobe was normal. The cysts showed characteristic columnar, ciliated, epithelium in its lining, with secondary infection and associated pneumonitis. The patient made an uneventful recovery following lobectomy, and to date has been free of pulmonary symptoms.

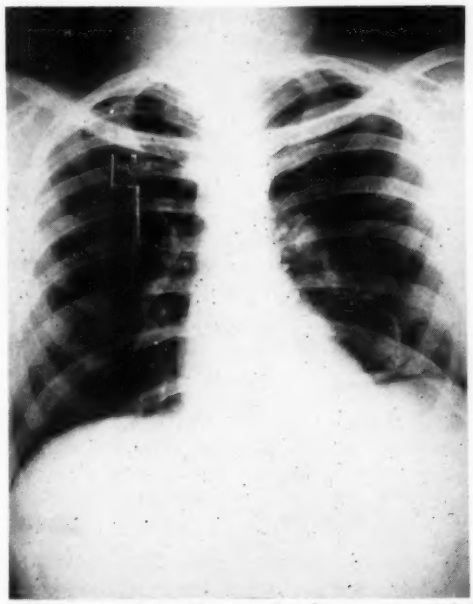

Figure 1.—Case No. 1, showing cysts of left lower lobe during relative asymptomatic stage, one year prior to surgery.

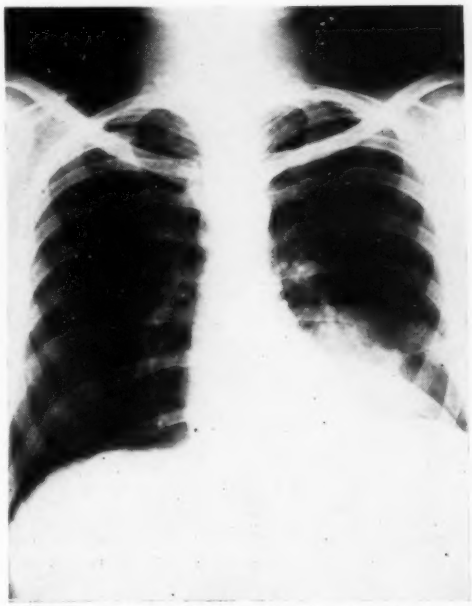

Figure 2.—Case No. 1, showing basal cyst of left lower lobe, filled with blood and purulent secretion.

Comment:

This is a classical case of unilobar congenital cystic disease that ran the entire gamut of complications, with progressive symptoms and disability. Earlier surgery would certainly have spared him two years of discomfort. There were no associated anomalies. Each cyst had a com-

municating bronchus, but repeated infection and thickening had all but occluded the bronchial orifice which explains the finding of the lower cyst tensely packed with undrained blood clot.

Case No. 2.—(See Figures 3 and 4) Patient is a 33 year old, white married female, who was perfectly well until October 1953, when she for the first time coughed up 1 to 2 ozs. of bright red blood. There was no associated febrile illness, cough or pain. An initial chest film was taken, and reported to be normal, though with slight prominence of the left hilum. The patient continued to raise small amounts of blood daily. Sputa studies were negative for acid fast, fungus disease, and neoplasm. During a bleeding episode in January 1954, she was bronchoscoped, and blood was seen to be coming from the superior segment orifice of the left lower lobe; the remaining examination was negative. With this information, oblique and Bucky films were taken of the suspected segment, with disclosure of what appeared to be a 3 cm. cystic lesion in the superior segment of the lower lobe. Hemoptysis continued and became progressive. At operation, on February 15, 1954, the patient was found to have a solitary 3 cm. congenital cyst of the superior segment of the left lower lobe with an aberrant bronchus patently connected to the left main stem. A segmental resection was carried out. The patient made an uneventful recovery, and has been symptom free since.

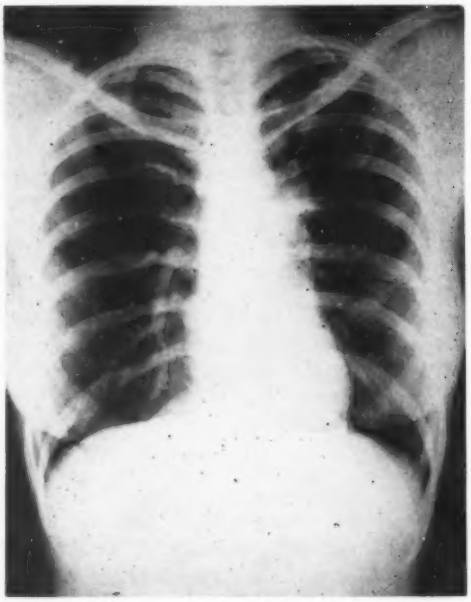

Figure 3.—Case No. 2, showing routine PA film at onset of hemoptysis, with only suggestive prominence of left hilum.

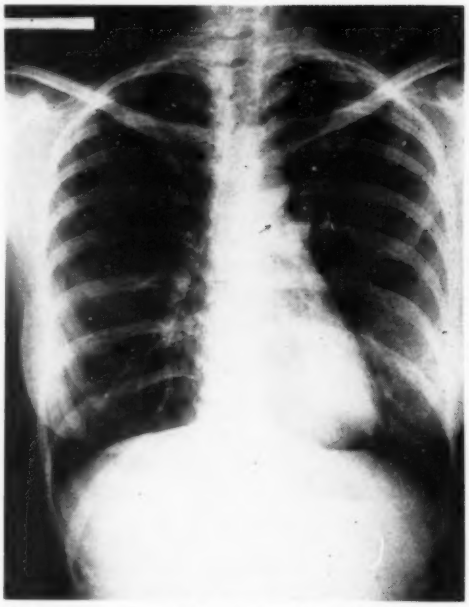

Figure 4.—Case No. 2, showing left hilar area one week after Figure 3, and revealing a 3 cm. cystic lesion in the superior segment of left lower lobe.

Comment:

This interesting case exemplifies the value of diagnostic bronchoscopy. It was by this means that the site of bleeding was localized, and a clue given that radiologically delineated the cyst. On section of the cyst, a myriad of thin-walled, fragile vessels were seen to course through the lining. There was some secondary infection, which presumably precipitated the bleeding.

Case No. 3.—(See Figure 5) Patient is a 48 year old, married white male, who entered the hospital with a six day history of cough, fever and right chest pain. His hospital temperature ranged up to 103 degrees. X-ray disclosed a homogeneous density of the lower half of the right lung field, with deviation of the trachae, to the left. It was felt that he had an underlying pneumonia and empyema. He showed no response to intensive antibiotic therapy. Attempted aspiration of the right plural space was unproductive. At bronchoscopy, after instillation of 1:10,000 aqueous Zephiran, there was sudden expectoration of about 500 ccs. of thick, purulent

Figure 5.—Case No. 3, showing the large filled cyst of the right middle lobe on admission, and again after bronchoscopy and lavage, when its cystic configuration became apparent.

secretion. A follow-up x-ray now disclosed a large cystic area that had apparently emptied its contents through the communicating bronchus. At surgery, eight days later, he was found to have a large, communicating, thick walled congenital cyst, completely replacing the medial segment of the right middle lobe. The cyst was about the size of a large grape-fruit, and crowded adjacent pulmonary segments. The cystic segment was excised, with complete recovery of the patient.

Comment:

This is a classical example of a large, communicating tension cyst that has become secondarily infected, with all the symptoms of pneumonia and picture of empyema. The value of bronchoscopy in emptying the filled cyst, and clarifying the underlying pathology is demonstrated.

SUMMARY

1. Congenital cystic disease of the lung is a not infrequent cause of pulmonary disability, often masquerading as carcinoma, tuberculosis, bronchiectasis, and unresolved pneumonia.

2. Secondary infection and bleeding are common complications.

3. Painstaking x-ray study and bronchoscopy are valuable diagnostic tools.

4. Surgical excision, when possible, is the ideal treatment.

5. Three representative cases are illustrated.

NOTE:

Case No. 3 is from the Thoracic Surgical Service of Boston City Hospital, Boston, Mass. (Dr. John W. Strieder, Chief of Service)

Cases Nos. 1 and 2 are from the surgical service of Our Lady of the Lake Sanatarium, Baton Rouge, La. (Drs. E. Lobrano and R. Coe, referring physicians).

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INFECTIOUS HEPATITIS IN CHILDREN *

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Infectious hepatitis in infants and young children, in contrast to the disease in adults, is not usually characterized by jaundice. Recognition of this fact has been relatively recent, and as a consequence, we are just learning about the incidence and duration of the disease, its spread, and the frequency of permanent liver damage following this infection in the pediatric age group. In older children the clinical picture and its severity becomes progressively more like that seen in adults. Expressed in another way, the severity of the disease is as a rule directly proportional to the age, ranging from mildly ill, nonjaundiced cases in infants to severely ill, jaundiced cases in older children. There are, of course, important exceptions to this general trend. Notable in the experience of one of us (C.G.) were two fatal icteric cases in young infants born to the same mother. The greatest incidence has been found between the ages of four and fourteen,[1] and has been related to the concentration of individuals in this age group in schools where lack of proper instruction in hygiene or limited sanitary facilities may contribute to the natural carelessness of children in their personal habits. In addition, the fact that many cases in the younger ages are not accompanied by jaundice, and, hence, may not be diagnosed or reported, suggests that the actual incidence in school-age children is considerably greater than recorded figures would seem to indicate.

ETIOLOGY AND MANNER OF SPREAD

These nonicteric cases greatly complicate the problem of controlling outbreaks of infectious hepatitis, whether in school or elsewhere, and probably are the source of a considerable number of secondary cases discovered in family groups. Drake[2] believes, from skin testing surveys, that this possibility is not as important as might be expected. Reports of other investigators have indicated that virus may be passed for long periods of time in the stools of patients who are not jaundiced. In any event, since it has been demonstrated that 35 per cent of adults over thirty years of age have built up an immunity,[3] infectious hepatitis must be considered as a disease to which a high proportion of young persons are exposed, and as a diagnostic possibility which must be borne constantly in mind by any physician who cares for children.

The viral etiology of infectious hepatitis has been established both by feeding experiments in human volunteers,[4] utilizing bacteria-free filtrates, and also by isolation and propagation of the viral agent in the amniotic fluid of embryonated eggs.[5] Cross infection and immunity studies have further established the difference between the viruses causing infectious hepatitis and serum hepatitis.[6] Recently a study in the St. Vincent's Orphanage in Chicago[3, 7-11] has furnished very convincing evidence that spread of the disease is accomplished—like typhoid—by the fecal-oral route. After controlling a well established outbreak of infectious hepatitis by conferring passive immunity with gamma globulin, Capps and his co-workers were able to prevent the appearance of further cases among new groups of student nurses entering training. Having established this fact, passive immunity was no longer provided, and for the following eight to ten month period new cases again appeared. Then aseptic technique, emphasizing careful handwashing, was instituted, and no further cases of hepatitis occurred among incoming nurses exposed to infected infants. In this connection it is interesting to note that no respiratory precautions were taken at any time. Stokes contends that at least a few cases would have been expected, even following the institution of aseptic technique, if respiratory spread

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were a significant possibility. However, because infectious hepatitis in children frequently begins with symptoms of upper respiratory infection, the possibility of respiratory spread has not yet been completely eliminated.

As would be expected in any disease spread primarily by the fecal-oral route, outbreaks have been reported which resulted from contaminated food,[12] milk,[13] and water.[14] It is probable, however, that more intimate personal contact explains a majority of cases. Finally, it should not be forgotten that the virus of infectious hepatitis can be recovered from the blood of infected individuals at least for a limited period of time,[3] and that as a result infection can be spread by the parenteral route, just as in the case of serum hepatitis.

DIFFICULTIES IN STUDY AS IN POLIOMYELITIS

In reviewing what is known, one is impressed by the effort and resources, both professional and otherwise, which have been devoted to the study of infectious hepatitis. Yet it is apparent that many important and fundamental questions remain unanswered. There are important reasons for this. First, and most important, there is no animal except man which can be infected with the virus of infectious hepatitis. Although limited experiments have been carried out utilizing human volunteers, this method does not permit extensive studies. Second, there is no diagnostic clinical or serological test or group of tests which can be used to establish a definite diagnosis. Finally, as mentioned previously, the demonstrated occurrence of nonicteric cases makes the task of studying epidemic outbreaks extremely difficult if not impossible. Much of the information we do have is based on a study of illnesses which are presumed to be infectious hepatitis because they have a suitable incubation period, because they present a typical sequence and type of symptomatology, because they are associated with suggestive abnormalities in laboratory tests, and because the occurrence of secondary cases among exposed, susceptible individuals is prevented by the use of gamma globulin.

Although the parallel is not complete, a study of infectious hepatitis presents many of the same difficulties that are encountered in dealing with poliomyelitis. There are undoubtedly many subclinical cases and most adults have acquired immunity without manifesting clinical forms of the disease. The difficulties in reaching more than a presumptive diagnosis are legion in both instances. The incidence of each disease has been increasing in spite of all efforts to combat it. In the case of infectious hepatitis, for example, during the first quarter of 1952, 6,286 cases were reported as compared to 8,216 cases during a similar period of 1953. During the first quarter of the current year there have been 17,120 cases—over twice as many as in the preceding year.[15] Part of this rise is undoubtedly due to more accurate diagnosis and reporting, but there is also probably an absolute increase. To carry the analogy with poliomyelitis further, treatment of both conditions is largely supportive, and, although a vaccine is now being tried in the prevention of poliomyelitis, we cannot describe a proved method for active immunization against either infection. With respect to passive immunity, gamma globulin is of limited, if any, value in poliomyelitis, but is very useful in infectious hepatitis. Finally, the viruses of both infectious hepatitis and poliomyelitis are passed in the stools of affected individuals for varying periods of time.

CLINICAL PICTURE AND DIAGNOSIS

Infectious hepatitis in infants, as in adults, begins after an average incubation period of twenty to thirty days.[6] The first symptoms are fever, irritability, and frequently, rhinitis. These are shortly followed by anorexia, nausea and vomiting, diarrhea, and an enlarged, tender liver, which at times is associated with severe abdominal pain and rigidity. Laboratory studies of liver function become abnormal during the prodromal period and frequently remain so for a considerable time after apparent clinical recovery.[16] Probably the earliest change is an increase in the direct

serum bilirubin reaction. This is observed even in infants who do not become clinically jaundiced and occurs before the total bilirubin becomes elevated in jaundiced cases. Another early change is the development of a positive thymol flocculation test. Since this reaction is also one of the last to revert to normal during recovery it is doubly useful as a clinical tool. Shortly after these tests become positive, the thymol turbidity and the cephalin-cholesterol flocculation also reveal abnormality, and still later, the total serum bilirubin may rise. These tests, along with urinary urobilinogen determination and an oral or intravenous hippuric acid excretion test, are recommended by Hepler[17] for evaluation of initial functional impairment in hepatitis and to follow progress of the disease process. In evaluating prolonged cases he recommends, in addition, the bromsulfalein test, serum albumin determinations, and measurement of prothrombin time in the presence of adequate vitamin K. In this connection, Stokes and Capps found, in studies at the St. Vincent's Orphanage, that in infants infectious hepatitis may follow a course of several months duration as determined by either clinical or laboratory studies. Furthermore, even after these infants appear well, and studies of liver function have returned to normal, they may continue to excrete the virus in their stools for many months. One infant in the St. Vincent's Orphanage study passed stools containing virus for at least fifteen months after apparent recovery. Of real interest, and in contrast to experience with adult cases, most of these infants appear to recover completely from such a prolonged illness without permanent damage to the liver.

The clinical picture in older children is essentially the same as that found in adults. Initially, there is a mild prodromal illness with fever lasting two to four days which is followed by an afebrile interval and apparent improvement for three to eight days. Jaundice then makes its appearance and is accompanied by an enlarging, tender liver. Typically, icterus becomes more intense for a period of three to seven days, at which time a crisis occurs with a subsequent rapid fall in serum bilirubin, re-appearance of bile in the stool, and prompt subsidence of nausea and liver pain. In the few cases which end fatally, the serum bilirubin continues to rise, nausea and vomiting persist, and twitching and somnolence rapidly progress to convulsions and coma. Death occurs soon thereafter, often within ten days of the onset of symptoms. Except in these fulminant cases, jaundice gradually fades during the one to three weeks following crisis. In this convalescent stage, the patient may appear clinically well but continues to have abnormal liver function studies for at least two to three more weeks.

MANAGEMENT

Although the fatality rate of infectious hepatitis is less than 1 per cent,[3] the prolonged period of morbidity makes it a disease to be universally respected. The proper management of it must accomplish two objectives: First, it must provide proper care for the individual; and, second, it must protect against spread of infection to susceptible contacts. The latter task is made particularly difficult by undiagnosed early or nonicteric cases as well as by unrecognized chronic cases which continue to excrete virus over long periods. Unfortunately, there is no biological, antibiotic or chemotherapeutic agent which affects the disease process directly. As a consequence, supportive therapy in general and complete bed rest in particular are of great importance. Not only does any exertion increase nausea and aggravate abdominal pain, but also activity will probably lead to a prolongation of the active phase of the disease and an increased incidence of permanent liver damage. In a study of 400 Navajo Indian children, Payne[19] kept 200 of them with active hepatitis at bed rest for several months. The other 200 children, who were thought to have had hepatitis but showed no evidence of activity on physical examination, were allowed to be active. At the end of the nine months, the percentage of enlarged livers, spider angiomata, and

abnormal liver function studies in the second group of children was greater than in the first. Some authors[20] recommend ambulation when appetite has returned and the bilirubin is normal, without waiting for the cephalin and thymol flocculation reactions to become negative. In contrast, Drake[2] urges that children be kept away from school until all liver function studies are normal, and believes that even then they should be observed carefully for fatigue, anorexia, or bilirubin in the urine.

Diet deserves major consideration as a supportive measure in the treatment of hepatitis, but in this instance palatability, rather than rigid control of specific types of food, must be the primary focus. For example, since fat tends to increase nausea and abdominal pain, it is logical to limit the amount ingested, but attempts to devise a fat-free diet yield such unappetizing results that they are justifiably abandoned in favor of a tempting well balanced diet. A liberal allowance of protein should be provided and usually is well accepted. The diet should supply an adequate amount of all vitamins, but unless food intake is seriously limited by anorexia or nausea, additional vitamins are unnecessary. Capps[21] has even suggested that an excessive vitamin intake may be harmful. Similarly, supplemental therapy with methionine and choline is unnecessary except in poorly nourished individuals or in those patients who for appreciable periods are unable to eat an adequate diet.[21] Antibiotics, of course, have no direct effect on the virus of infectious hepatitis itself, and do not significantly alter the course of the disease. Both Drake[2] and Capps,[21] however, recommend a brief course of aureomycin in severely ill children on the hypothesis that perhaps by cutting down the number of intestinal bacteria, the liver is relieved of part of its burden in detoxifying the products of bacterial metabolism. As in the case of any liver disease, the response of patients to barbiturates is both exaggerated and prolonged. Nevertheless, the cautious administration of small doses of phenobarbital is indicated for control of marked restlessness.

The results of treatment with adrenocorticotropic hormone (ACTH) and adrenal cortical extract (ACE) have been spotty, conflicting, and in general, disappointing. Webster[22] reported good results when ACE was used in the treatment of several forms of severe liver disease, including infectious hepatitis, but was not able to discern any remarkable beneficial effect of ACTH.[23] In addition, a high incidence of side effects was associated with its use. Both Colbert[24] and Klatekin[19] reported general clinical improvement, with a decrease in anorexia and pruritis as well as a fall in serum bilirubin with the use of ACTH, but they also pointed out the necessity for continuing therapy until recovery is almost complete in order to prevent relapse when therapy is discontinued. While some authors[21] feel that neither ACTH nor cortisone is indicated, under any circumstances, at least it can be stated that they should not be used except in severely ill or comatose children.

Probably the most important phase of the management of infectious hepatitis, whether in a community or family, is the prevention of secondary cases. In view of the nature of the disease, this is not easily accomplished. The long incubation period of twenty to thirty days makes it impractical to isolate contacts, and tends to dissipate the attitude of watchful waiting engendered by known exposure. The virus has been demonstrated in the stools of patients by the time of the first vague prodromal symptoms,[11] and quite possibly it is present even before this. As a result, an obvious reservoir of virus exists in the excreta of recognized cases and in the stools of infants with nonicteric, often undiagnosed disease. This latter group of patients is epidemiologically quite important and can be considered as at least temporary carriers. They offer a probable explanation for the discrepancy between the frequency of naturally acquired immunity in the general population as judged by skin testing and a medical history of

jaundice.[18] Unfortunately, there is no method by which this "carrier state" can be either terminated or prevented. There are, however, means at our disposal of preventing the spread of infectious hepatitis. In the first place, temporary protection conferred by gamma globulin has been conclusively demonstrated [25, 26] and finds its greatest usefulness in preventing secondary cases in family groups and closed institutions.[1] Effective levels of passive immunity are obtained with doses of gamma globulin as small as 0.01 cc. per pound of body weight if given at least seven days before the expected onset of jaundice. Protection can be expected to persist for as long as six weeks. Isolation of active cases, proper facilities and instruction in sanitation, and aseptic techniques have their obvious applications, especially in institutions with stable populations.

A recent, unexpected observation has suggested the possibility that a vaccine may be developed which will offer active immunity against infectious hepatitis. When an epidemic of infectious hepatitis occurred in a children's house of correction in which a portion of the population had been previously skin tested [28] using irradiated amniotic fluid from embryonated eggs infected with the hepatitis virus, Drake [18] found that there were 5 cases of hepatitis among 320 tested children, or a rate of 1.6 per cent as contrasted to 112 cases among the 825 children who had not been tested—a rate of 13.6 per cent. This suggests that a considerable number of the skin tested group had acquired immunity.

In considering the marked rise in the number of cases of hepatitis with jaundice in the older age groups, and the high incidence of this disease in children from five to fourteen years of age, it is interesting to speculate further as to the similarity between poliomyelitis and infectious hepatitis. Is this increased incidence related to the relatively high standards of cleanliness and hygiene which prevail today, and which in turn tend to prevent the acquisition of active immunity in infancy through repeated subclinical infections? Solid active immunity to infectious hepatitis is certainly important since it has been variously estimated that up to 10 per cent of the general population harbor the virus and thus serve as possible sources for exposure of susceptibles.

SUMMARY

Infectious hepatitis is a disease of prime importance to any physician caring for children. Its greatest incidence is in individuals between the ages of five and fourteen years, but of even greater importance, it frequently assumes a nonicteric form in infants and young children, the vague symptomatology of which is particularly difficult to recognize. Even though the clinical course in these young people is often prolonged, they seldom suffer permanent liver damage. The fact that they excrete virus in their stools over a period of many months constitutes a difficult problem in preventive medicine which has not yet been solved. The management of infectious hepatitis in children, as in adults, is almost entirely symptomatic. Prevention of the disease is possible by means of careful attention to personal hygiene and by the use of gamma globulin.

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TREATMENT OF OSTEOARTHRITIS OF THE HIP JOINT*

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Osteoarthritis, commonly known as degenerative arthritis involving the hip joints, or malum coxae senilis, comprises a gradually increasing percentage of the practice of the orthopedic surgeon. Undoubtedly, this increase can be explained largely on the basis of the increase in life expectancy and the admirable attempts of the medical profession as a whole to make elderly patients more useful and comfortable (Figure 1).

ETIOLOGY

In standing and in walking, at every step the entire weight of the body is carried through the hip joints. Imperfect development, previous disease, injury, and the normal degenerative effects of age all contribute to impairment of function of this important joint. Among the underlying etiologic factors, many of which occur in childhood or adolescence and lead to degenerative changes in adult life, may be mentioned those due to imperfect development of the hip, such as congenital coxa vara or congenital dislocation or subluxation of the hip with associated dysplasia of the acetabulum, and those due to adolescent epiphyseal disturbances, such as coxa plana or slipping of the upper femoral epiphysis. Other predisposing causes include pyogenic infection of the joint or complications of infectious diseases such as scarlet fever. Still other degenerative changes develop after direct trauma to the joint (Figure 2), such as aseptic necrosis of the head of the femur following fractures of the head or intracapsular fractures of the neck of the femur, traumatic dislocations or fracture dislocations involving portions of the acetabulum. The latter are becoming more frequent as the speed and power of our motor vehicles increase. Lastly, the daily wear and tear, and the normal result of aging in major weight bearing joints, universally present after the age of 40 years, are important etiologic factors.

PATHOLOGY

The clinical manifestations of this disease closely parallel the pathologic changes that develop as the disease progresses (Figure 3). In the early stage, the femoral head shows some subchondral cysts and some marginal sclerosis with diminution of the articular space. Later, there develop marginal exostoses as a result of

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Figure 1. Severe osteoarthritis following fracture dislocation of hip in 37 year old male. (A) Immediately following injury. (B) 17 years later.

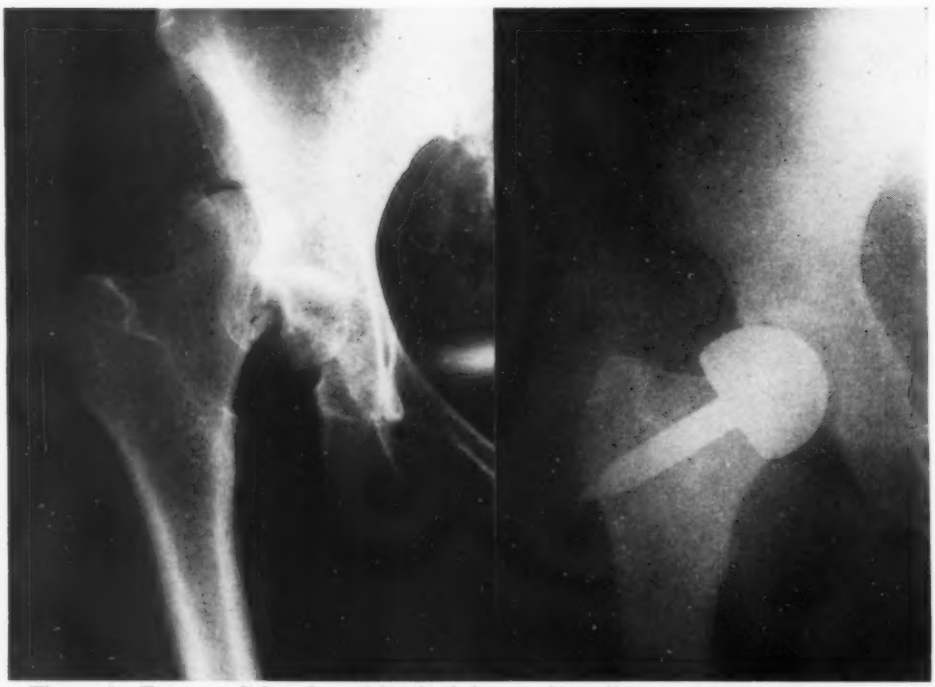

Figure 2. Fracture dislocation of head of femur. Immediate treatment with replacement prosthesis in attempt to prevent changes such as those demonstrated in Figure 1.

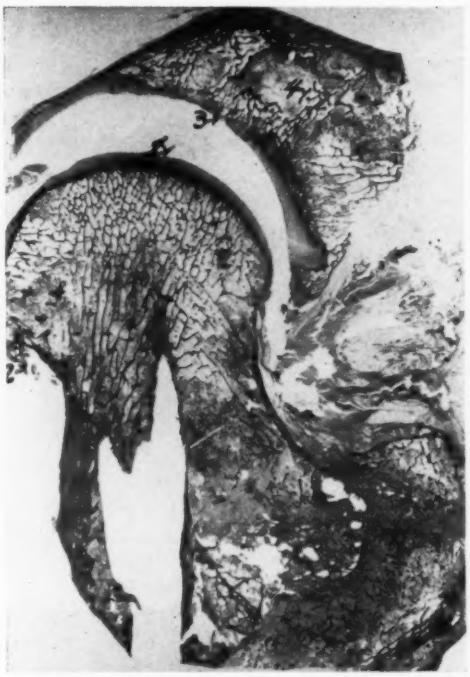

Figure 3. Autopsy specimen following fracture of base of femoral neck in 80 year old patient. Shows typical pathologic changes of osteoarthritis: (1) Degeneration of cartilage of head; (2) Lipping of cartilaginous margins; (3) erosion and thinning of weight bearing cartilage of acetabulum; (4) areas commonly known as subchondral cysts are areas of bone necrosis with replacement by edematous fibrous tissue.

proliferation of new bone about the femoral head and margin of the acetabulum. The cartilage becomes irregular and worn away over the head and sclerosis of the head as well as the adjacent acetabulum becomes evident. Finally, there are evidences of severe deformity. There may be some outward and actual subluxation of the head with a mushroom appearance over the neck of the bone.

CLINICAL MANIFESTATIONS

In the early stages the principal complaint is pain on weight bearing with increased stiffness of the hip. Pain is felt chiefly on motion and is relieved by rest. It may extend down the thigh to the knee. Gradually, muscle spasm develops and the hip assumes a characteristic position, primarily fixation in outward rotation, flexion and adduction. Movement of the hip joint is restricted, particularly in internal rotation, and pain and limping become progressively worse. As flexion and adduction increase, the patient assumes a peculiar gait not unlike a gluteus medius limp because he must swing the body to the opposite side in order to clear the adducted hip from the floor in walking. At this stage it may be necessary for the patient to use a cane or a crutch in order to relieve the pain of weight bearing sufficiently to remain ambulatory. (Figure 4)

TREATMENT

Preventive Measures. — Ghormley[1] recently listed three factors which he believed might be effective in reducing the incidence of degenerative disease of the hip. The first is eradication of acute infection of the hip in infancy and childhood as a result of the availability of chemotherapeutic and antibiotic agents. The second is better care of patients with congenital dislocation or dysplasia of the hip because of earlier recognition and more satisfactory treatment. The third factor is earlier recognition and more effective treatment of slipping of the femoral capital epiphysis and Legg Perthe's disease. In addition, better initial care of traumatic dislocations and acute fractures of the head of the femur and acetabulum would certainly help prevent disabling degenerative changes in later life resulting in traumatic arthritis (Figure 6).

Conservative Measures. — Osteoarthritis of the hip causes pain, instability and deformity or limitation of motion. Of these, pain is the most important consideration in determining whether conservative or operative treatment is indicated in the individual case. A young person with an unstable painful hip is more seriously handicapped than an elderly person, since he is forced to pursue a sedentary occupation. Older individuals can often be relatively happy and self sufficient if they can be relieved of pain by conservative means, even being willing to use a crutch or cane. Many of them will adjust to the deformity or limitation of motion or instability if only they can be

Figure 4. (A) Degenerative arthritis in 54 year old male following septic arthritis at age 21. (B) End result, four years postoperatively; (C) Twelve years after arthrodesis patient has no pain, works on feet 8 to 10 hours daily; required cane previous to operation.

Figure 5. Osteoarthritis following slipped upper femoral epiphysis. End result seven years after arthrodesis at age of 56. Works daily in hat shop.

Figure 6. Vitallium cup arthroplasty following partial fibrous ankylosis of hip. Result of septic arthritis as a child. One year after operation 75% of hip motion.

rid of the pain. The ability of the orthopedic surgeon to judge the amount of subjective pain, and the ability of the patient to adjust to his disability are probably the greatest factors in determining whether conservative or operative measures are required.

Much can be accomplished in the early stages by limitation of activity, weight reduction, frequent rest periods, use of recumbency with traction to overcome early deformities, use of crutches or complete bed rest during an acute exacerbation, and generous use of aspirin. Physical therapeutic measures, such as heat, massage and exercises, are of secondary importance. Occasionally, use of a lumbosacral corset will be of material aid in improving posture and holding the head of the femur snugly against an uninvolved portion of the acetabulum.

Injection of procaine into the obturator nerve and the posterior sensory supply to the capsule from the small branches of the sciatic nerve, advocated principally by Steindler,[2] has, in our experience as well as that of many others, resulted in only transient relief of pain.

Since the advent of hydrocortone reports of its efficiency in relieving symptoms in osteoarthritic hips have varied considerably. Hollander[3] reported failures in 53 per cent of 77 hips treated in this way. He attributed these failures principally to difficulty in penetrating the hip joint capsule and also the fact that seldom can fluid be aspirated to assure the operator that the needle is within the joint. He used an anterior approach just lateral to the femoral artery. The usual dose is 1.5 cc. or 37.5 mg. On the other hand, Reynolds and Ramsey[4] reported satisfactory results in 14 of 18 patients who had received 28 injections of hydrocortone into the hip for degenerative arthritis. A result is considered satisfactory if at least 50 per cent of the pain is relieved for a period of three weeks.

Roentgen ray therapy, although popular for short periods of time, has been discarded by most competent radiologists.

Operative Measures.—Increasing severity of the disease and intolerable pain are the principal indications for operative treatment. Denervation of the hip joint, popularized by Obletz and associates,[5] has proved unreliable in our experience. This leaves only three other surgical possibilities: (1) arthrodesis, (2) osteotomy, and (3) arthroplasty.

Of all procedures for the control of pain arthrodesis is undoubtedly the most reliable. It is ideal for a young active man with unilateral disease who works outdoors. It is the surest method of eliminating pain as well as restoring suitable position of the joint. However, it has the disadvantage of placing some additional stress upon the lumbosacral joint, and for this reason many surgeons consider extensive arthritic changes in the lumbar spine a contraindication to its performance. In addition, prolonged postoperative immobilization is required in order to ensure fusion of the denuded articular surfaces. However, once fusion is obtained, the patient is assured a permanent, painless extremity in good position on which he can bear almost unrestricted weight, unless there is considerable shortening but this produces little noticeable limp. We have considered this the procedure of choice for unilateral involvement at any age.

We have treated 17 consecutive osteoarthritic hips by arthrodesis. Seven patients were under the age of 50 years, 5 in the sixth decade and 5 in the seventh decade of life. All patients have been followed from four to ten years. Postoperative infection prevented fusion in one patient and one patient died after three years. The other 15, who all required crutches preoperatively are not only able to walk without crutches, but are able to resume their former occupation or an equivalent one. Only 2 complain of mild low back pain after considerable activity, and only 2 have appreciable limitation of motion of the knee due to immobilization.

Most of the several standard operative technics for securing arthrodesis of the adult hip include removal of the articular cartilage from the head and acetabulum,

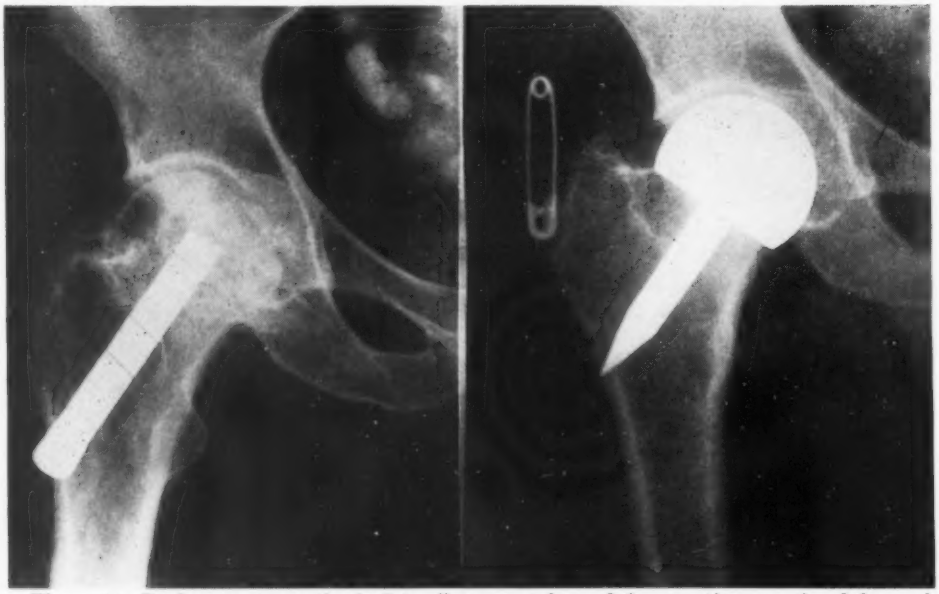

Figure 7. Replacement prosthesis "stem" type performed for aseptic necrosis of femoral head three years after internal fixation for fracture of femoral neck.

fixation with some type of metal, usually a Smith-Petersen nail driven through the neck and head into the thick portion of the acetabulum with additional fixation screws at right angles to the nail, and use of additional bone, usually from the ilium, to supplement fusion. The accepted position is approximately 30 degrees of flexion, not more than 5 degrees of abduction and slight external rotation. Usually, at least four to six months are required to obtain radiologic evidence of fusion.

The second method, *osteotomy*, moves the weight bearing line of the femoral shaft toward the midline of the body and corrects faulty position due to adduction flexion contracture with its secondary strain on the lumbar spine. Steindler[2] considered suitable candidates for osteo-

Figure 8. Replacement prosthesis "intramedullary" type performed for nonunion of femoral neck fracture.

tomy those who have no nocturnal pain and have at least some range of adduction and flexion without pain, because, after osteotomy, the flexed hip is extended, the adducted hip is abducted and the range of motion in these directions must be painless if the result is to be satisfactory. This operation not only corrects the deformity but brings a different or uninvolved portion of the femoral head in contact with the acetabulum to carry the body weight. This method has been employed extensively in England, particularly by McFarland.[6] It has not enjoyed wide popularity in this country because its advantages over arthroplasty or arthrodesis have not seemed convincing.

Arthroplasty (Figure 9) has the advantage of preserving or increasing motion but patients must be carefully selected. It is definitely indicated in patients with bilateral disease, especially if motion has been obliterated, since it is most desirable to secure motion in at least one hip. For a successful result it is necessary to have sufficient strength and muscles about the joint to move the hip under weight bearing conditions postoperatively. Moreover, the patient must be willing to cooperate in a long program of exercise to restore atrophic muscles to their normal capacity and particularly overweight individuals must be willing to demonstrate a genuine desire to improve their condition by losing weight before the operation is attempted.

In general arthroplasties have several disadvantages. Some instability results, not all pain is eliminated in many cases and the extremity frequently cannot withstand constant weight bearing so that the patient is forced to sedentary employment.

Two methods of arthroplasty are in general use. One, the vitallium cup or mold arthroplasty, was popularized by Smith-Petersen.[7] The other method is prosthetic replacement, either of the stem type originated by Judet[8] in which the head only is replaced, or of the intramedullary type which replaces the head and all or part of the neck with fixation in the femoral shaft.

The vitallium cup arthroplasty has been in use since 1936. It requires a high degree of technical skill on the part of the operator to reshape the head and acetabulum. In the hands of Smith-Petersen and his co-workers, Badgley, Ghormley and others, a high percentage of good results has been obtained.

The replacement type of arthroplasty, which has been in use only the past four years, has not yet been perfected. Many complications have been reported. The acrylic and nylon materials have been largely supplemented by metallic materials, either vitallium or stainless steel, and

Figure 9. Arthrodesis and arthroplasty performed for relief of severe bilateral osteoarthritis in patient 56 years of age.

the mechanical effect of the prosthesis upon weight bearing and stress reaction of the supporting neck and shaft of the femur has not been satisfactorily determined, because of the short period of time that the method has been employed. Another outstanding disadvantage is that after sacrifice of the head or head and neck, if the method is unsuccessful because of infection or other complications and the prosthesis is removed, it is extremely difficult to reconstruct or arthrodese the hip as a secondary procedure because of the loss of bony substance.

SUMMARY

Treatment of osteoarthritis of the hip is conservative or operative depending upon the amount of pain and progressive disability which the patient experiences. The surgeon's ability to evaluate the patient's complaints as well as to estimate the patient's ability to carry on with certain restrictions in activity is of paramount importance in abandoning conservative treatment for operative treatment. There are three operative procedures for correction of osteoarthritis of the hip: arthrodesis, subtrochanteric osteotomy or arthroplasty. Again, the surgeon's ability to select the correct procedure for the particular patient, based on his knowledge of the requirements of the patient and his own personal experience with these methods, is the key to successful results.

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SOME PRACTICAL ASPECTS ON THE DIETARY TREATMENT OF DIABETES MELLITUS

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An adequate diet is a part of the treatment of every patient with diabetes mellitus. The conventional methods used in the past to attain this have been complicated; the foods have been of limited variety and have required painstaking weighing. In many respects the philosophy of dietary instruction has overemphasized the importance of food restriction. This negative approach has resulted in an emotional maladjustment in certain patients. The purpose of this paper is to discuss present day trends to promote emotional well-being and sociological adjustment of the individual, while supplying him with a balanced diet.

Calculation of a diet formula based on the standard weight of the patient is the first step in providing adequate nutrition. The total caloric intake should vary from 25 to 50 calories per kilograms of body weight depending upon age and occupation. An adult patient doing office work or housework requires about 30 calories per kilogram of body weight. The twenty-four hour total caloric formula can thus be calculated. Obese patients should be put on reduction diets varying from 1200 to 1500 calories (or 1000 calories in extreme cases) and so regulated that they lose 1 to 2 pounds per week.

After the diet formula is computed the patient is given instructions which will make it possible for him to translate these calories into the proper type of food. A relatively new diet has been formulated jointly by the American Dietetic Association, American Diabetic Association, and Diabetes Section of the United States Public Health Service.[1] One of the chief advantages of this "ADA Diet" is its simplicity.[2] It is not the purpose here to review this new food plan in detail, but

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a few of the important refinements will be discussed.

Table 1 shows the caloric formula for the 6 meal plans. They provide variation in intake and distribution of protein, fat, and carbohydrate, calories which will fit most patients. Plans 5 and 6 provide a quart rather than a pint of milk and are designed primarily for children.

TABLE 1
CALORIC LEVELS OF SAMPLE MEAL PLANS

MEAL PLAN	CARBOHYDRATE	PROTEIN	FAT	ENERGY
	gm.	*gm.*	*gm.*	*calories*
1	125	60	50	1200
2	150	70	70	1500
3	180	80	80	1800
4	220	90	100	2200
5 *	180	80	80	1800
6 *	250	100	130	2600

* Planned particularly for children.

The last step in translating these calories to actual foodstuffs is done by the use of so-called food exchanges. Each of six food exchanges represents a measured amount of milk, meat, fruit, vegetable, bread or fat (Table 2). A booklet given to the patient lists the various foods, grouped according to their composition into these six exchanges.

The vegetable exchange list (Table 3) shows several revolutionary changes from the older diets. All vegetables containing 6 per cent or less carbohydrates are grouped under A. These can be used in unlimited quantities raw or as much as 1 cup when cooked. This furnishes a useful source of bulk food to satisfy the appetites of young boys or of patients on reduction diets. Such a method obviates the confusion and time previously spent weighing and adding 1, 3 and 6 per cent vegetables which formerly were listed separately. Vegetable exchange B indicates vegetables which contain more sugar and should be used in measured amounts.

The bread exchange includes foods containing 15 grams of carbohydrate, such as vegetables of high sugar content. In marked contrast to older diabetic diets is the presence of pure starch foods such as potatoes, rice, and spaghetti. For the children, as well as some adults, cake and ice cream may be used. (Table 4)

The "ADA Diet" thus provides every variety of food which the nondiabetic patient normally eats, except sugar. Substitutes for this form of carbohydrate, such as sucryl and crystallose, are real improvements over saccharin with its bitter after taste. Diabetic candy and soft drinks, such as ginger ale, orange and root beer made with a sugar substitute, are also now on the market. These are useful especially in children to help eliminate the feeling of being "different". It must be emphasized that these are substitute items. The patient should not only adhere to his designated diet but know it so well that he can eat in public gatherings in complete ease.

The treatment of patients with diabetes mellitus who cannot tolerate a solid diet deserves some attention. Such occasions arise during illness and before and after operations and necessitate the use of a liquid diet or parenteral fluid administration. For short term illnesses, the protein and fat content of the diet can be eliminated. The food given in twenty-four hours should adhere to the total amount of carbohydrate which the patient received prior to the illness. A liquid diet can be simply compiled using liquid exchanges from the ADA Diet. As an example, an eggnog with 2 teaspoonfuls sugar, one cup of orange juice and a slice of toast with butter provides about 60 gms. of carbohydrates and 500 calories. A comparable meal three times a day can adequately supply a patient with this regular intake of carbohydrates and calories.

Parenteral fluids should be administered by using glucose. A 5 per cent solution supplies 50 grams of carbohydrate per liter and a 10 per cent solution supplies 100 grams. An amount commensurate with the regular intake of carbohydrate can be given. In long term illnesses

TABLE 2

ADA MEAL PLAN No. 5

For.....

Use Only As Your Doctor Prescribes

Carbohydrate 180 grams, Protein 30 grams, Fat 30 grams Calories 1800

This meal plan has been prepared to use with the Meal Planning Booklet.

YOUR FOOD FOR THE DAY

Amount	Kind of Food	Choose From
1 quart	Milk	List 1
Any Amount	Vegetable Exchanges A	List 2A
1	Vegetable Exchanges B	List 2B
3	Fruit Exchanges	List 3
6	Bread Exchanges	List 4
5	Meat Exchanges	List 5
3	Fat Exchanges	List 6

Divide this food at follows:

YOUR MEAL PLAN

Breakfast:
- 1 Fruit Exchange from List 3
- 1 Meat Exchange from List 5
- 1 Bread Exchange from List 4
- 1 Fat Exchange from List 6
- 1 cup Milk from List 1

Lunch or Supper:
- 1 Meat Exchange from List 5
- 2 Bread Exchanges from List 4
- Vegetable from List 2A (any amount)
- 1 Fruit Exchange from List 3
- 1 Fat Exchang from List 6
- 1 cup Milk from List 1

Dinner or Main Meal:
- 3 Meat Exchanges from List 5
- 2 Bread Exchanges from List 4
- Vegetable from List 2A (any amount)
- 1 Vegetable Exchange from List 2B
- 1 Fruit Exchange from List 3
- 1 Fat Exchange from List 6
- 1 cup Milk from List 1

Bedtime Meal:
- 1 cup Milk from List 1
- 1 Bread Exchange from List 4

SAMPLE MENUS

These Menus Show Some Of The Ways Exchange Lists Can be Used To Vary The Meal Plan The Exchange Lists And Recipes May Be Found In The Meal Planning Booklet

Breakfast:
- Orange Juice—½ cup
- Egg—1
- Toast—1 slice
- Butter—1 teaspoon
- Milk—1 cup (8 ounces)

Lunch or Supper:
- Cheese Sandwich
  (Cheese—1 ounce, Bread—2 slices, Butter—1 teaspoon)
- Lettuce and Tomato Salad
  Zero Salad Dressing (recipe page 17)
- Apple—1 small
- Milk—1 cup (8 ounces)

Dinner:
- Hamburg Patties—3 ounces
- Mashed Potato—½ cup
- Carrots—½ cup Spinach
- Bread—1 slice
- Butter—1 teaspoon
- Banana—½ small
- Milk—1 cup (8 ounces)

Bedtime:
- Milk—1 cup (8 ounces)
- Graham Crackers—2

Breakfast:
- Prunes—2 medium
- Cereal, cooked—½ cup
- Boiled Egg—1
- Butter—1 teaspoon
- Milk—1 cup (8 ounces)

Lunch or Supper:
- Bologna—1 slice
- Radishes, Celery
- Potato Salad (A)—½ cup (recipe page 18)
- Bread—1 slice Butter—1 teaspoon
- Grapefruit—½
- Milk—1 cup (8 ounces)

Dinner:
- Tomato Juice—small glass
- Roast Lamb—3 ounces
- Steamed Rice—½ cup
- Green Peas—½ cup
- Mixed Green Salad
  Zero Salad Dressing (recipe page 18)
- Bread—1 slice Butter—1 teaspoon
- Pineapple Gelatin (recipe page 15)
- Milk—1 cup (8 ounces)

Bedtime:
- Milk—1 cup (8 ounces)
- Soda Crackers—3

TABLE 3
VEGETABLE EXCHANGES

VEGETABLE EXCHANGES A Contain little Carbohydrate, Protein, or Calories		VEGETABLE EXCHANGES B Contain 7 grams Carbohydrate, 2 grams Protein and 35 Calories	
Asparagus	Lettuce	Beets	
Broccoli	Mushrooms	Carrots	
Brussels Sprouts	Okra	Onions	
Cabbage	Pepper	Peas, Green	1 exchange — ½ cup
Cauliflower	Radishes	Pumpkin	
Celery	Sauerkraut	Rutabagas	
Chicory	Young String Beans	Squash, Winter	
Cucumbers	Summer Squash	Turnip	
Escarole	Tomatoes		
Eggplant	Watercress		
Greens			

TABLE 4
BREAD EXCHANGES—LIST 4

One Bread Exchange contains 15 grams Carbohydrate, 2 grams Protein and 70 Calories.

The list shows the different amounts of food for 1 Bread Exchange.

	Amount to Use		Amount to Use
Bread	1 slice	Flour	2½ Tablespoons
Biscuit, Roll (2″ diameter)	1	Vegetables:	
Muffin (2″ diameter)	1	Beans & Peas, dried, cooked (lima, navy, split pea cowpeas, etc.)	½ cup
Cornbread (1½″ cube)	1	Baked beans, no pork	¼ cup
Cereals, cooked	½ cup	Corn	⅓ cup
Dry, flake & puff types	¼ cup	Pop Corn	1 cup
Rice, Grits, cooked	½ cup	Parsnips	⅔ cup
Spaghetti, Noodles, cooked	½ cup	Potatoes, white	1 small
Macaroni, etc., cooked	½ cup	Potatoes, white, mashed	½ cup
Crackers, graham (2½″ sq.)	2	Potatoes, sweet or Yams	¼ cup
Oysters (½ cup)	20	Sponge Cake, plain (1½″ cube)	1
Saltines (2″ sq.)	5	Ice cream	½ cup
Soda (2½″ sq.)	3	(Omit 2 fat exchanges)	
Round, Thin (1½″)	6		

where parenteral fluids are desirable amino acids are used to supply the protein. The use of parenteral emulsions, which are high in calories, is now in the investigative stage. To date these preparations have been found to be safe, but an adequate method of preventing rancidity has not been perfected.

SUMMARY

1. Recent trends of the dietary treatment of diabetes mellitus are discussed.

2. Emphasis is placed on methods which simplify the diet and allow greater food variation.

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ROENTGENOLOGIC ASPECTS OF GASTRIC CANCER *

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The seriousness of cancer of the stomach is reflected in its high incidence among neoplastic diseases and its continued high mortality rate.[1, 2] The annual number of deaths from this disease in the United States has reached about 40,000,[3] and in Louisiana the annual death rate is approximately 16 per 100,000 population.[4]

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The imperative necessity for recognition of cancer of the stomach while it is a localized lesion is thus apparent. In most cases this obligation rests with the physician who does the roentgenologic examination. Only by early recognition is there any real hope of increasing the present extremely low survival rates.

It is generally agreed that radiologic examination offers the simplest, most available, and probably most accurate method for diagnosis.[5-7] With this in mind and also with a view to correlating the roentgenologic observations, we undertook a review of the cases of gastric cancer at the Ochsner Clinic and Ochsner Foundation Hospital.

MATERIAL

A diagnosis of cancer of the stomach was established 290 times in 150,000 consecutive new patient registrations. Eleven of these were excluded because they were sarcomas; these will be considered in a separate report. Of the 279 carcinomas, 32 had to be excluded because of incomplete roentgenologic study or because of the unavailability of the films. The remaining 247 cases form the basis for this report. The roentgenograms and fluoroscopic reports of all but 9 of these were reviewed by us. In these 9, a report by a competent radiologist who performed the examination was used, since the films were not available for restudy.

ANALYSIS OF CASES

Area of Involvement: Contrary to the usual concept that the lesser curvature of the stomach is more frequently involved than the greater curvature,[6] in our series the distribution at these two sites was almost the same (Fig. 1), being 59.5 per cent in the former and 60.5 per cent in the latter. These figures indicate, of course, involvement of both curvatures in many patients, which is almost inevitable in advanced disease.

Our data support the general opinion that the lower half of the stomach is more frequently the site of malignancy than the upper half.[8] The cardia and fundus were the sites of disease in 25.1 and 18.6 per cent, respectively, whereas the gastric

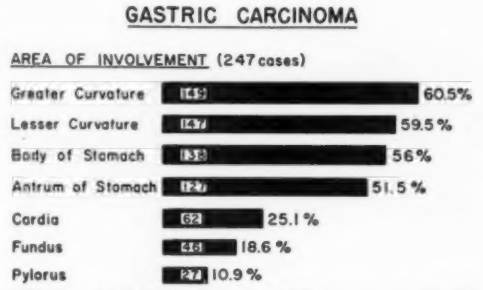

Figure 1. Distribution of 247 cases of gastric carcinoma according to area of involvement.

antrum and pyloric areas were involved in 62.4 per cent of cases. From a roentgenologic diagnostic viewpoint, it is fortunate that gastric cancer has a predilection for the distal half of the stomach, since this portion is more easily examined because of its lower position in relation to the thoracic rib cage and its more anterior location in the abdominal cavity. Both factors favor manipulation at fluoroscopy and compression film technic, methods which are accentuated in modern diagnosis.[8, 9]

Roentgenographic Observations: A *constant filling defect* in the outline of the stomach, caused by neoplastic growth above the surrounding mucosa, which has been considered the fundamental roentgenologic sign in gastric carcinoma,[9-11] was observed in 60.5 per cent of our cases (Fig. 2). When the stomach is filled with barium mixture, a large fungating mass (Fig. 3) may stand out in bold relief along the gastric contour. Demonstration of smaller lesions requires a more

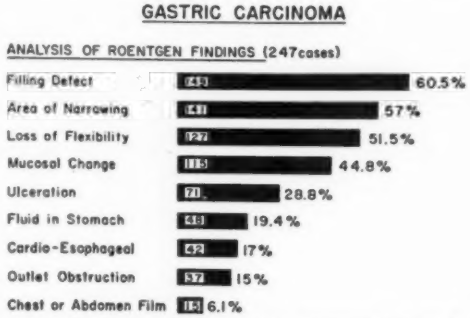

Figure 2. Roentgenographic observations in 247 cases of gastric carcinoma.

Figure 3. Filling defect on greater curvature of stomach.

Figure 4. Area of narrowing, lower half of stomach.

subtle approach, as they may be observed with only a small amount of barium coating the gastric mucosa. Kirklin,[12] as Carman before him, stressed the imperative necessity of alert, methodical and understanding fluoroscopic study of the gastric mucosa. The examination must be done after only one or two swallows of the barium mixture. Shanks [13] emphasized this by quoting with approval Berg's aphorism, "The less you give the more you see." The desirability of visualizing the stomach in the supine and prone positions, as well as in the erect one, has also been emphasized.[9]

Narrowing of the gastric outline (Fig. 4) and *loss of flexibility* of the stomach wall, which were noted in slightly more than one half the cases in this series, are usually related. They result when the tumor infiltrates the wall of the stomach, stiffening it and preventing normal flexibility and distensibility. In advanced lesions the whole stomach may be involved, producing a "leather bottle stomach" or linitis plastica. A less extensive lesion may be limited to one part of the stomach, or even one curvature alone. Disordered gastric peristalsis may at times be associated with these infiltrative changes in the wall of the stomach. Detecting alterations in the peristaltic waves is of debatable value, and in our experience has been of only limited diagnostic aid.

Changes in the appearance of the gastric mucosa were present in slightly less than half the cases in the present series. These produce two quite different appearances. In one type the mucosa appears flattened and "washed-out," with loss of the normal pliable folds. As seen in profile, there is a fine uneven irregularity, suggestive of small mucosal ulcerations. Kirklin,[12] who described this as a "granular internal relief," considered it one of the most reliable signs of scirrhous carcinoma. In the other, less common type of mucosal change the folds are larger than average and show evident irregularity and deformity. These changes result from infiltration of the mucosa by the cancer, which produces a stiffening of the mucosa. It is this stiffening and irregular deformity of the mucosal folds which usually permit differentiation from "gastritis." Like-

wise, in our experience, associated changes are almost always present to aid in the diagnosis. Rarely does gastric carcinoma produce only enlargement of the mucosal folds.

Ulceration as a roentgenologic feature in gastric cancer is a complex and interesting problem. The controversy continues as to whether or not benign gastric ulcers frequently become malignant. The current trend seems to be toward the belief that ulcerating carcinoma is generally cancer from the onset.[14-17] Roentgenographic evidence of ulceration (Fig. 5)

Figure 5. Ulcerating carcinoma on greater curvature side of gastric antrum. Note so-called "meniscus sign".

was found in 28.8 per cent of our series. Of these, 48 were less than 2 cm. in size, 32 being on the lesser curvature and 18 on the greater curvature. One showed ulceration on both curvatures. Of the 26 ulcerations greater than 2 cm. in size, 11 were on the lesser curvature (3 in antrum, 8 in body of stomach), 14 on the greater curvature (4 in antrum, 10 in body), and one on the posterior aspect of the fundus of the stomach.

Much has been written about differentiation of benign and *malignant ulcers*. Factors favoring a diagnosis of malignant ulcer are: older age of patient, nontenderness over visualized ulcer, associated mass, location of ulcer in prepyloric area or greater curvature, larger size of ulcer, irregular ulcer crater, stiffening or alteration of adjacent mucosa, inset of ulcer in relation to normal mucosal outline, and the "meniscus sign" of Carman and Kirklin due to the raised neoplastic margin of the malignant ulcer. When the barium coated stomach is compressed, with the malignant ulcer in profile, a translucent zone about the base of the ulcer which has the appearance of a meniscus is visualized; when seen *en face* this zone appears as a halo about the ulceration.[12, 18] No one of these observations in itself is definitive. Failure to respond promptly to adequate medical therapy is as important as any other factor. Evaluation of this healing response by serial roentgenologic study is not always easy. We agree with Shanks [7] that "if, after careful survey of all the x-ray features, there is doubt (and there often is), the radiologist should not hesitate to say so . . . Surgical exploration may be necessary. It is far better to cure a simple ulcer by partial gastrectomy than to miss a carcinoma."

It is interesting that 3 of our cases occurred in patients with a known "gastric ulcer" which had been treated medically at the Ochsner Clinic for periods of one to six months; in 4 others the "gastric ulcers" had been treated by other physicians for periods of two to twelve months. It is this feature about gastric ulceration —that 10 to 20 per cent of those considered originally benign are proved subsequently to be malignant—which has led Ochsner and Blalock [19] and other surgeons to advocate prompt excision for ulcerating disease of the stomach.

Fluid in the stomach at the beginning of the examination, despite fasting, was observed in 19.4 per cent of these patients. Such observation should arouse suspicion of some obstructive lesion at the gastric

outlet. This is not always indicative of obstruction; however, as some patients will have ingested fluid without admitting it. In others, the flow of gastric secretion may be excessive or pylorospasm may be responsible. Although it may alert the examiner to possible outlet obstruction, the presence of excessive fluid seriously hampers examination of the gastric mucosa. If the gastric contents cannot be aspirated before the examination, it is usually wise to reexamine the patient on a subsequent day.

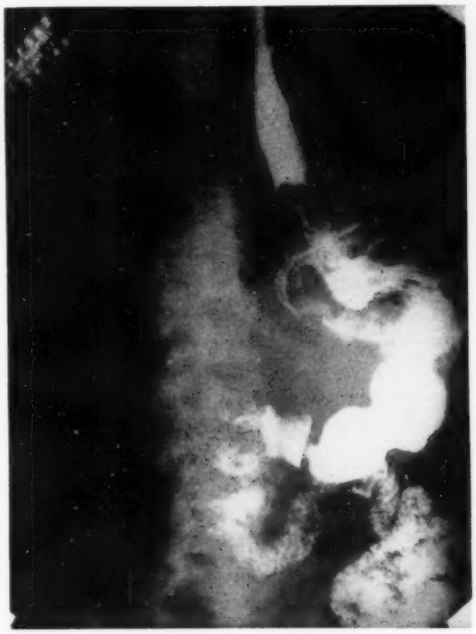

Figure 6. Cardio-esophageal type of tumor.

In 17 per cent of our patients the *cardio-esophageal* area (Fig. 6) was involved. This is the same percentage reported by Pack,[20] who added, "In our experience most cancers involving the lower esophagus are found to be of stomach origin." Cancer originating in the stomach readily spreads to the esophagus. Reporting that 22 per cent of resected carcinomas of the stomach were located in the region of the cardia, Sherman[21] stressed the features of mucosal alteration and infiltration with rigidity or contour change.

Outlet obstruction (Fig. 7) was present in 15 per cent of the 247 patients. Retained fluids or food may obscure the features which would suggest neoplastic obstruction, such as irregular narrowing, filling defect, and loss of flexibility. It is, therefore, extremely important to examine the stomach when it is empty. An annular neoplastic lesion at the outlet may readily mimic a chronic stenosing gastric or pyloric ulcer. Visualization of a large stomach with flexible walls does not mean that the obstructing lesion is necessarily benign.

Figure 7. Outlet obstruction with dilated stomach.

In 6 per cent of these 247 patients, or 33 per cent of the cardio-esophageal cancers the tumor could be seen in plain roentgenograms of the chest or abdomen. This is dependent upon the tumor being cast into relief by air in the stomach. Such tumors must of necessity be fungating masses and must almost always be in the fundus or at the cardia of the stomach. This is an interesting and important observation, indicating that the gastric air bubble should be carefully scrutinized in all films of the chest and abdomen. In reviewing 68 cases of cancer at the cardia of the stomach, Kirklin and Gilbertson[22]

found that about half of these could be seen as a projecting soft tissue mass in the gastric air bubble. As Templeton [9] pointed out, the tumor is more clearly defined by coating it with barium and then visualizing it in the air bubble.

Negative Roentgenograms: Cases in which a significant gastric lesion was not discerned were included in this study in an attempt to detect faults in the examination. There were 13 such cases in this series; this represents a known diagnostic error of 5.2 per cent. In order to avoid bias in favor of the radiologist, fairly vigorous standards were established. Thus, included among these 13 cases are 1 in which carcinoma of the pancreas was the radiologic diagnosis and 2 in which carcinoma of the esophagus was correctly diagnosed but an associated gastric neoplasm was not detected.

Worthy of note is that in 6 of these 13 cases a positive diagnosis was made on re-examination—2 within one week, 3 more within one year, and 1 three years later. In 2 additional cases, in which the films showed positive evidence of disease, which was overlooked, conspicuous lapses of personal judgment must be accepted. Thorough and conscientious work will reduce errors to a minimum.

A gastric cancer which is missed on initial roentgenographic examination rarely remains undetected for a prolonged period. In this series one case was classified as "missed," which showed extensive disease three years later. This may be too critical a criterion, for these lesions are by nature progressive, and often rapidly so. In the usual case, re-examination is soon demanded, or else the associated findings are so compelling that exploratory laparatomy is undertaken despite the negative radiologic report.

Five Year Survival: Twelve patients survived at least five years after surgical excision of the carcinoma, all of whom had lesions of the distal half of the stomach. Two showed outlet obstruction, 8 involvement of the gastric antrum, and 3 the body of the stomach. In 6 the type of lesion was a marginal filling defect due to the fungating tumor, and in 5 it was an ulcerating lesion. Two of the ulcerations were less than 2 cm. in diameter; 3 were greater than 2 cm. In 1 the stomach was considered normal roentgenographically.

Types of Roentgenographic Pattern: There are five fairly distinct roentgenographic patterns which cancer of the stomach may exhibit. Based upon these gross manifestations, a satisfactory roentgenologic classification is possible (Table 1).

TABLE 1
TYPES OF GASTRIC CARCINOMA IN 247 CASES

TYPE	CASES	%
Cardio-esophageal	42	17
Fungating	149	61
Ulcerating	71	29
Infiltrating	127	52
Obstructing	37	15

In advanced disease combined types are seen.

At the upper orifice of the stomach, where the esophagus is readily involved, cardio-esophageal carcinoma is found. This type accounted for 17 per cent of the 247 cases in our series. Disease which manifests itself chiefly by exuberant growth protruding into the gastric lumen produces a fungating or polypoid lesion, noted in 61 per cent of these series. Ulcerating carcinoma was seen in 29 per cent. Infiltrating lesions result in thickening and stiffening of the gastric wall, with obliteration or deformity of the mucosal pattern, often called scirrhous carcinoma. We observed these changes in 52 per cent of the patients. In his review of gastric neoplasms, Stout [14] listed these lesions with the following percentages: fungating 22.7 per cent, ulcerating 27.4 per cent, infiltrating 12.9 per cent, with 37 per cent showing mixed characteristics.

At the lower gastric orifice, carcinoma may result in outlet obstruction. This was found in 15 per cent of our series.

CONCLUSIONS

1. Cancer of the stomach is a common neoplasm, with an insidious onset and a high mortality.

2. Roentgenologic examination of the

stomach is at present the easiest, most available and probably most accurate method of diagnosis.

3. The examination must be methodical and thorough, with careful fluoroscopy and compression spot-films in addition to conventional films.

4. There are five roentgenographic patterns of cancer of the stomach: cardio-esophageal, fungating, ulcerating, infiltrating, and outlet obstruction.

5. If the results of the roentgenologic examination are inconclusive, prompt re-examination is recommended. If doubt still remains, gastroscopy and exploratory laparotomy deserve immediate consideration.

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POTENTIAL DANGER IN THE TREATMENT OF ASTHMA WITH ARSENIC CONTAINING MIXTURES

Arsenic has long been known as a dangerous drug and as an insidious poison. In recent years, an arsenic containing mixture has been advocated by certain practitioners for the treatment of asthma. These prescriptions contain inorganic arsenic in the form of Fowler's solution (potassium arsenite). One reported prescription for this type of mixture is as follows:

Fowler's solution	6.00 cc.
Potassium iodide	6.5 Gm.
Tincture digitalis	2.33 cc.
Saccharin	0.065 Gm.
Elixir of phenobarbital	q.s. 120 cc.

The dosage of Fowler's solution (potassium arsenite) in the prescription is approximately 3 minims per teaspoon. In certain other mixtures there may be three times as much arsenic, and in one reported case the patient was taking 20 to 28 drops of Fowler's solution three times a day.

Various reports have been made indicating that these asthma remedies have been shipped through the mails over an extended period, during which the patient was not seen or examined for the possible toxic effects the arsenic might have produced. Sollman [1] has stated: "Inorganic arsenic is capricious, unpredictable, and uncontrollable both as to good and harm; but the harm is more certain and generally more frequent than the good."

Inorganic arsenic and organic arsenic are both capable of acute and chronic poisoning with far reaching effect. Symptoms appear with reference to all organ systems and may produce a variable number of disease pictures. The development is insidious and fatigue and loss of energy may be the only early manifestations. In the nervous system, there may be blindness, sensory disturbances, pains in the joints, sensory and motor paralyses, possibly with muscle atrophy and hypesthesia. In the gastrointestinal system, there may be jaundice, signs of toxic hepatitis, diarrhea, anorexia, emaciation, and nausea. Acute hepatitis may be fatal. The effects produced in the skin are numerous, such as edema, hyperhidrosis, flushing, acne, eczema, scaling of the skin, keratoses of the palms and soles, and a pigmentary condition. The toxic effect on the bone marrow may produce aplastic anemia.

Inorganic arsenic is absorbed promptly through the intestinal tract, part of which is excreted in the urine, and other portions go to the epithelial and, to a less extent, the endothelial structures. Arsenic may be

found in the urine many months after the cessation of its ingestion. It is stated that it may be found in the hair and in the nails for an indefinite number of years after ingestion.

A recent article by Hansen-Pruss [2] gives the report of a study of 17 adults, ranging in age from 33 to 65. These patients received inorganic arsenic in the form of an asthma mixture, such as the "Gay Treatment," or possibly, variations of this formula. The individuals studied received this treatment over a period ranging from two weeks to two and one-half years. The time interval elapsed between the last administration of inorganic arsenic and the time they came to Duke Hospital under this investigator ranged anywhere from three weeks to three years. Fifteen of the 17 patients gave a positive test for arsenic when their hair was examined. Seven of the 17 had a urinary content of arsenic which exceeded the normal. Skin lesions varied from a mild and transient erythrodermia to an irreversible hyperkeratosis involving mainly the extensor surfaces of the extremities and the soles of the feet. Two of the group had abnormal biochemical tests compatible with liver damage. A majority of the group developed some form of gastrointestinal complaint. Symptoms in this field appeared two to six weeks after the treatment had been started with arsenic.

In addition to the reports by this author, there are other articles in the last several years [3-6] showing the serious toxic effects from the arsenic contained in this treatment. Consideration of the known facts about the toxicology of arsenic and the data presented in these several reports indicates that the tolerance, as has been known for generations, for arsenic is variable. It also indicates that in certain individuals there is an extreme susceptibility to the poisonous effects of arsenic, and that in such individuals serious toxic degenerations are set up with the inorganic form in a subtle fashion, just as much as with the organic form when organic compounds of arsenic were being used in the treatment of syphilis.

Among the conclusions offered by the author of the most recent article,[2] it is stated that there is no authenticated therapeutic reason why inorganic arsenic should be effective in asthma, and that its administration is often deleterious. It is further stated that the ingestion of inorganic arsenic compounds produces immediate or delayed skin reactions in many individuals. The latter appear usually after three to eight years, are irreversible, and at times are carcinogenic. It is also concluded by this author that the 17 patients reported on were not benefitted by this type of treatment.

It is quite clear then that arsenic is an insidious poison and is to be used only with great care under adequate supervision. It is also to be remembered by physicians that an individual suffering from asthma and presenting bizarre and unusual symptoms may be one who has had this type of treatment. It becomes necessary, therefore, to suspect the toxic effects of arsenic in any asthma patient.

1. Sollman, T.: A Manual of Pharmacology and Its Applications to Therapeutics and Toxicology, ed. 7, Philadelphia, W. B. Saunders Co., 1948, p. 1132.

2. Hansen-Pruss, O. C.: Arsenic in the treatment of asthma, Southern M. J. 48:270 (March) 1955.

3. Pelner, Louis, and Waldman, Samuel: Toxic hepatitis caused by prolonged inorganic arsenic intake: Studies on liver dysfunction—VII, New York State J. Med. 51:2511, (Nov. 1) 1951.

4. Prickman, Louis E., and Millikan, Clark H.: Hemorrhagic encephalopathy during arsenic therapy for asthma, J.A.M.A. 152:1711 (Aug. 29) 1953.

5. Pascher, Frances, and Wolf, Jack: Cutaneous sequelae following treatment of bronchial asthma with inorganic arsenic; report of two cases, J.A.M.A. 148:734 (March 1) 1952.

6. Silver, Alfred S. and Wainman, Philip L.: Chronic arsenic poisoning following use of an asthma remedy, J.A.M.A. 150:584 (Oct. 11) 1952.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

REPORT OF PRESIDENT

Soon after adjournment of the House of Delegates last May, it became necessary to focus attention on the proceedings of the State Legislature. It was gratifying to see at one committee hearing over two hundred physicians from all parts of the state. This is the kind of assistance that our legislative committee must have if we expect to accomplish anything constructive in legislation for better health of the community. Taking everything into consideration, the results were good. However, for the first time in history, the chiropractic issue came to the floor of the House of Representatives as a proposed Constitutional Amendment. Though this was soundly defeated, it is evident that sentiment in favor of licensure for this cult is becoming stronger every day. If we desire to continue this fight, and continue we must, a sound program must be developed in the Louisiana State Medical Society to work every day of every year instead of just a few weeks before the session of the Legislature.

The matter of medical testimony in the courts is important. In a court of law when a physician is called as a witness he not only testifies for a patient and his lawyer, he represents a great profession and by his performance which is given in public, he can reflect credit or discredit on the whole medical profession. Court records bear witness to the fact that a few members of this noble profession at times appear in court unprepared, some are inexperienced and incompetent, and only a few, thank God, are dishonest.

Malpractice cases have increased, the amount of money granted by the courts has also increased tremendously. The attorney for the Medical Defense Committee handled sixteen cases for the Society in 1954, and is prosecuting at present approximately fifty chiropractic injunction suits for the Louisiana State Board of Medical Examiners. He advises there were numerous other malpractice claims handled by insurance companies and not reported to the Society. Many of the answers to prevention of further increase in this insidious hazard may be found in the list of rules for prevention of malpractice suits which was mailed to every member of the Louisiana State Medical Society in February of this year. Proper indoctrination of new members by component societies, establishing grievance committees and discussion of fees with patients before treatment, also application of the Golden Rule will help materially. A poor medical record is all a plaintiff's attorney needs to obtain a quick, easy and lucrative judgment.

At the present time the analysis of the survey in the state in regard to malpractice insurance rates and the possibility of obtaining blanket coverage has not been completed. As to the possibility of coverage from the national level, this is remote because rates vary so markedly throughout the states.

In legal circles there are murmurs of a whitewash being given in our own investigations and reports by our committees. Each case must be thoroughly and honestly investigated and evaluated at local level. The inquiry should not originate with bias or prejudice in either direction, nor should the recommendation from the parish society committee bear false witness.

Uniform insurance blanks are near to becoming reality and this will come from AMA level where it belongs.

Membership and component society activity is improving because of the more realistic approach to our need for better public relations. There is much to be accomplished along these lines. It is impossible to accomplish what is necessary in this day and time with entire dependence on the voluntary part-time effort of the officers and the committees no matter how proficient or diligent they may be.

Activities of component societies lag perceptibly in some areas because of poor geographical distribution. For instance in the Seventh District, doctors of two parishes on the eastern end of the district rarely attend the Seventh District meeting. In the Third District a similar situation exists. If agreeable with the majority of members concerned, it will be proposed that a ninth district be added.

The physical facilities of the State Society office are totally inadequate. The Louisiana State Medical Society certainly should not be dependent for their office space forever.

All through the above one can denote a thread of much to be accomplished. There is reflected the need for something in organization that this Medical Society does not have. There has long been the need for closer contact between the Louisiana State Medical Society office and the individual member. Because the House of Delegates requested the Executive Committee to work out a plan for employment of public relations counsel and as a result of attendance at Public Relations Conferences in Chicago and Miami, a special committee was asked to work out the details. Every delegate has a copy of this report or will have by the time the 1955 House of Delegates convenes. A considerable number of component organizations

have been briefed on the details. The reason this was done is that if this program is accepted it will be necessary to raise the dues again.

A full time layman trained in public relations can assist the component societies by setting up contact meetings of parish society officers with a public official and his physician before and after his election to an office concerned with the health of the community. This elected representative will know of the attitude and program of organized medicine before the opposition has developed its program. The public relations director will be able to be present at meetings of the component societies for advice and assistance in arranging membership canvassing, TV and radio programs, development of indoctrination and grievance committee programs. It is visualized that the function of this office will be varied and will bring about better organization and understanding. There should be no authority as to policy vested in this office. Both the Council on Medical Service and Public Relations, and the Public Policy and Legislation Committee will be well served by this employee; overlapping activities of these two groups can thus be avoided.

Recommendations

1. Increase dues ten dollars yearly to develop public relations department.

2. Increase dues five dollars yearly to establish a fund for a domicile.

3. Make provision for a ninth district if no major objection from members so concerned.

4. Send copies of abstracted Minutes of House of Delegates to presidents of component societies for their information with suggestion that they be read to the members.

5. Encourage the practice of physicians insisting that prescriptions be filled at pharmacies of patient's own choice (a step forward in keeping peace and harmony resulting from personal communication with representative of Louisiana Pharmaceutical Association).

6. Serious thought be given to the problem of integration. The Louisiana State Medical Society must be prepared for any eventuality.

7. Resolution to AMA with copies to other state societies, requesting medical schools to intensify medico-legal courses, with emphasis on medical testimony and prevention of malpractice cases.

8. President of each component society be urged to request a lawyer to address each society on malpractice prevention and medical testimony once each year.

9. Programs related to School Health, Mental Health, Rural Health and Health Council work should continue to be of interest and concern to the physician and organized medicine.

WALTER MOSS, M. D., President

RECOMMENDATION FOR ADMINISTRATION OF THE SALK VACCINE

The National Foundation for Infantile Paralysis announced today that it would follow the recommendation of Dr. Jonas E. Salk that for the year 1955 vaccine be administered in two doses, separated by an interval of two to four weeks, followed by a third dose not earlier than seven months later but before the 1956 poliomyelitis season.

Dr. Hart E. Van Riper, Medical Director of the National Foundation, stated that this decision was made on the recommendation of the Vaccine Advisory Committee of the National Foundation, consisting of Dr. Thomas E. Rivers, Chairman, Director of the Rockefeller Institute for Medical Research; Dr. David E. Price, Assistant Surgeon General, United States Public Health Service; Dr. Richard E. Shope, member, the Rockefeller Institute for Medical Research; Dr. E. L. Stebbins, Director, School of Hygiene and Public Health, The Johns Hopkins University; Dr. Norman Topping, Vice President in charge of Medical Affairs, University of Pennsylvania, and Dr. Thomas Turner, Professor of Microbiology, School of Hygiene and Public Health, The Johns Hopkins University.

The pharmaceutical houses manufacturing the Salk vaccine agreed to supply the National Foundation sufficient vaccine to immunize all children in the first and second grades throughout the United States and its territories, and all children who participated in the 1954 field trials but who did not receive vaccine. The total number of these children is estimated to be 9,000,000.

Reducing the number of doses that can be given before the oncoming epidemic season from three to two, as recommended by Dr. Salk, will require the use of approximately 9,000,000 less doses of 1 cc. each than would otherwise have been necessary. These 9,000,000 doses will remain with the pharmaceutical houses for distribution through commercial channels and will provide the first two doses for an additional 4,500,000 children immediately.

Since Dr. Salk's recommendation is that the third dose be given not earlier than seven months later, it will be impossible for the National Foundation to offer that dose to the same children, for obvious reasons. This fact, however, will be compensated by the immediate availability through commercial channels of the first two doses for an additional four and a half million children because of the National Foundation's following Dr. Salk's recommendations.

The National Foundation will offer a booster dose to the 500,000 children who actually received vaccine in the 1954 field trials, which was also recommended by Dr. Salk.

Dr. Van Riper also announced that the National Foundation was requesting all pharmaceutical houses to ship immediately sufficient vaccine to cover the first dose.

CONFERENCE ON VACCINE ALLOCATION: AMA TO PARTICIPATE

Following announcement that the Salk vaccine was effective in preventing poliomyelitis, these developments occurred in Washington:

1. President Eisenhower ordered a national survey to determine a fair voluntary method for allocating supplies, with a general conference of interested associations scheduled on April 22.

2. The White House announced that full information on Dr. Salk's vaccine would be turned over to every nation with which the U. S. has diplomatic relations, including a number behind the Iron Curtain. This was regarded mainly as a humanitarian gesture, as data on the vaccine and the field tests have been made public.

3. The Department of Health, Education, and Welfare officially licensed six firms to manufacture and sell the product, thereby leaving decisions on allocation and price in the hands of private industry, public health departments, and physicians.

Under instructions from President Eisenhower, Secretary Hobby of the Department of Health, Education, and Welfare has begun a survey to determine the best way to insure the "fair distribution" of Salk poliomyelitis vaccine to children in greatest need of it. The President said he was interested in setting up a voluntary system for allocating the product until expanding production facilities can meet the demands.

Mrs. Hobby already has held conferences with medical officers of federal agencies to get basic information and their recommendations. Next step is contact with representatives of the AMA and other groups, as spokesmen for physicians, and with representatives of all other interests involved in the vaccine question, including pharmaceutical houses. A meeting was tentatively scheduled for Friday, April 22, when these groups would meet with government officials.

Pres. Eisenhower asked that the discussions cover two points, "to insure that channels of distribution of the vaccine be kept open and that equal opportunity of treatment is given every section of the country." He said he wanted a mechanism set up whereby the doctors and the drug industry would "see to it that no child who should receive the vaccine will be denied it."

The six firms licensed by the Department of Health, Education, and Welfare to make and distribute the vaccine are: Cutter Laboratories, Berkeley, Calif., Eli Lilly Co., Indianapolis, Ind., Parke-Davis & Co., Detroit, Mich., Pittman-Moore Co., Zionville, Ind., Sharp and Dohme, Philadelphia, Pa., and Wyeth Laboratories, Inc., Marietta, Pa.

INFORMATION ON INSECTICIDE POISONINGS REQUESTED

A special grant has been made to the Department of Pathology, Louisiana State University School of Medicine, to investigate the mechanism of action of anticholinesterase insecticides and the therapy of poisoning resulting from excessive exposure. The widespread use of Parathion and TEPP (tetraethyl pyrophosphate) in agriculture, industry and the home is causing an increased number of accidental exposures. Our interest encompasses clinical observations as well as studies on fatal cases at autopsy.

If a case of poisoning by any of the anticholinesterase insecticides comes to your attention, it would be appreciated greatly if you would allow us to obtain information on the case. Because of the rarity of this condition and certain unusual aspects of therapy we are prepared, when requested, to give recommendations as to treatment in a surviving patient. We are also prepared to send an investigative team at our expense.

Telephone calls to report cases and request information will be accepted at any time, day or night, to:

Dr. Stanley H. Durlacher, New Orleans,
Franklin 4141, collect
Department of Pathology
Louisiana State University
School of Medicine

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MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

ANNUAL MEETING LOUISIANA HEART ASSOCIATION

The Louisiana Heart Association will hold its annual meeting on June 16, 1955 in the Louisiana State University Medical School Auditorium, New Orleans. The scientific session will begin at 2:00 p. m., followed by the first annual assembly of Louisiana Heart Association members at 4:00 p. m. A public forum on heart disease will be held at 8:00 p. m., sponsored by the Louisiana State University Medical School and the Louisiana Heart Association.

CAUSES AND CURES TOLD FOR BACKACHES

Standing erect may help distinguish man from the animals but it certainly lets him in for a lot of trouble, a doctor of public health said recently.

Dr. James A. Tobey, Newton, Conn., said the human skeleton "is not particularly well adapted to the upright position," except for a short time when a man is young, "lean, lithe, and buoyant." The rest of his life, he is particularly prone to backache and other ills which might not occur "if he ran on all fours," Dr. Tobey said in the February issue of Today's Health magazine, published by the American Medical Association.

Instead of actually standing erect, many people "slouch and sag, droop and sway," sometimes because of fat or cares and emotional stresses. Other factors are extra high heels, occupational hazards, later stages of pregnancy, strains, and disease. Faulty body mechanics is one of the most common causes, one of the most important, and usually the easiest to correct.

The one-third of each day spent in bed may be a factor, he said. Mattresses should be flat and even, not too soft, and free of "hollows, lumps and slants." But a good mattress is not enough. There are 57 other possible causes of backache, Dr. Tobey said.

CRIPPLED CHILDREN'S HOSPITAL COMPLETED

The $1,750,000 Crippled Children's Hospital at New Orleans has been completed and has received its first patients. The facility, which will provide convalescent and rehabilitation care in beautiful modern surroundings, is open to patients from the entire state. Initial patients are from Lafayette, Crowley and New Orleans.

Children undergo surgery or pass the acute stage of their illness in general hospitals, then are transferred to the Crippled Children's hospital. After they no longer require hospitalization, they may receive follow up care through the hospital's extra large, air-conditioned outpatient department.

The hospital has 50 beds for inpatients, x-ray, laboratory, examination rooms, physical therapy cubicles, a Hubbard tub, whirlpool bath, therapeutic pool, plaster cast facilities, a large, specially equipped gymnasium, occupational therapy workshop, music room, two classrooms and other facilities.

Care of the patients may be financed through the state health department, National Foundation for Infantile Paralysis, National Society for Crippled Children and Adults and other health organizations, insurance plans, or by the patient, according to his ability to pay. All children up to the age of 21, regardless of creed, color or ability to pay may be admitted, but they must be handicapped children for convalescent or rehabilitation care, and in no case are they to be alcoholic, insane, custodial whether due to mental or physical handicaps, narcotics addicts or have a communicable disease.

Further information may be obtained from the Administrator, Crippled Children's Hospital, 200 Henry Clay Avenue, New Orleans.

BOSTON CLINICAL MEETING AMERICAN MEDICAL ASSOCIATION

November 29-December 2, 1955

All persons who desire a place on the lecture program at the Boston Clinical Meeting of the American Medical Association are urged to communicate immediately with the Chairman of the Program Committee—Theodore L. Badger, M. D., c/o Massachusetts Medical Society, 22 The Fenway, Boston 15.

Applications for space in the Scientific Exhibit are now available and will be sent on request. Exhibits will supplement the lectures as far as possible, and should portray subjects of a broad general interest. Requests for application should be sent to the Secretary, Council on Scientific Assembly, American Medical Association, 535 N. Dearborn St., Chicago 10, Illinois.

ANNUAL ASSEMBLY IN OTOLARYNGOLOGY

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from September 19 through October 1, 1955. This Assembly will consist of two parts.

Part I. September 19 through September 24, 1955, will be devoted to surgical anatomy of the head and neck, fundamental principles of neck surgery and histopathology of the ear, nose and throat. This week will be under the personal direction of Maurice F. Snitman, M. D.

Part II. September 26 through October 1, 1955, will be devoted entirely to lectures and panel discussion of advancements in otolaryngology. The chairman of this section will be Emanuel M. Skolnik, M. D.

Registration is optional for one or both weeks.

TENTH ANNUAL SCHERING AWARD COMPETITION OPENS FOR NATION'S MEDICAL STUDENTS

The tenth annual Schering Award competition for medical students has opened, it was announced by Robert W. Burlew, M. D., chairman of the award committee. Its aim is to encourage medical writing and exploration of current research literature.

Titles of three subjects on which American and Canadian students are invited to submit papers this year are: Current Concepts in the Management of Osteoporosis; Prevention and Treatment of Blood Transfusion Reactions; and Recent Trends in the Clinical Use of Adrenocortical Steroids.

Both a $500 first prize and a second one of $250 will be awarded for each of the three subjects. Outstanding authorities in each field will select the winners. Special recognition in the form of a professional gift will be given each student submitting a meritorious paper.

Deadline for entry forms specifying the choice of title is July 1st. Manuscripts should be mailed not later than October 1st. Students may compete individually or cooperatively in teams.

Information and instructions for the award competition are available from the Schering Award Committee, 60 Orange Street, Bloomfield, N.J.

PHYSICIANS OFFER IDEAS ON AUTO SAFETY

Physicians are continuing their search for a way to reduce highway accident deaths.

Two physicians, in letters published in a recent (Feb. 5) Journal of the American Medical Association, today debated the merits of auto safety belts and agreed that belts alone are not the entire answer. They said over-all automobile design must be improved and other safety measures developed.

The A.M.A.'s House of Delegates, meeting in San Francisco in June, passed a resolution calling for wider use of safety belts, and recommending that auto manufacturers emphasize safety in all car designs.

Dr. John H. Schaefer, Los Angeles, said he believes that auto seat belts as presently designed will save no lives. He said a device that would distribute force evenly over a large area of the body and would yield in absorbing this force would be "valuable."

He also said that placing seats facing backward and building them high enough to support the head would be a "logical answer to the problem of motor car collision, but I doubt that the public would accept it."

BOOK REVIEWS

Hernia: The Pathologic Anatomy of the More Common Hernias and Their Anatomic Repair; by Chester B. McVay, M. D., Springfield, Illinois, Charles C Thomas, Pp. 40, Price $4.75.

This is an excellent review of the hernia problem in general and the more common hernias. It is well written and contains numerous self-explanatory semi-diagrammatic illustrations. It gives a clear concise picture of the anatomical approaches and is recommended for use by surgeons and students.

CHARLES C. ABBOTT, M. D.

Neck Dissections; by James Barrett Brown, M. D., and Frank McDowell, M. D., Springfield, Ill., Charles C Thomas, 1954, Pp. 163, Price $7.50.

This book discusses the indications, limitations, complications, and usefulness of neck dissections. It presents techniques of various types of neck dissections. It also discusses the treatment of lip and oropharyngeal lesions. The discussion of preoperative and postoperative treatment is good. There are many pictorial case presentations and a fine series of colored drawings of neck dissections. It is recommended to anyone interested in cancer of the lip, oropharynx, and neck dissections.

CHARLES C. ABBOTT, M. D.

Fundamentals of Neuropathology; by William Brooks Dublin, M. D., Springfield, Illinois, Charles C Thomas, 1954, Pp. 685, $18.50.

Of the several textbooks recently published on the fundamentals of neuropathology, Dr. Dublin has written one of the most practical and readable. The purpose of this book is to emphasize neuropathology in terms of general pathology. The clinico-pathological correlations are excellent and make the book good reading for the general pathologist as well as for the clinician. Pertinent associated pathological findings outside the central nervous system are skilfully blended in the text. The laboratory techniques are discussed in the beginning of the book thus orienting the reader in the interpretation of later histologic description. Laboratory staining methods which are likely to be of use in a general pathology laboratory are emphasized. Occasionally, the author is ambiguous in his description of a few of the rarer diseases, but this does not detract from the general usefulness of the book. The sections of the book are well organized. The gross and microscopic descriptions of the pathological lesions are adequate. The illustrations are excellent. The development of the nervous system and the histogenesis of its several cell types are well reviewed.

This is a practical textbook of neuropathology and the author is to be commended for his clear and concise presentation of a difficult subject. In the reviewer's opinion the bibliography would have served a better purpose had it been placed at the end of the appropriate chapters. The book is printed on good paper.

This textbook fills a long recognized need and merits little adverse criticism. It can be read by clinician and pathologist alike with considerable benefit to both.

JOHN H. DENT, M. D.

Review of Medical Microbiology; by Ernest Jawetz, Ph. D., M. D., Joseph L. Melnick, Ph. D., and Edward A. Adelberg, Ph. D., Los Altos, California, Lange Medical Publications, 1954. Illus. Pp. 360. Price $4.50.

This modestly priced, paper-bound textbook is intended primarily for the medical student, house officer, and practicing physician. It is clearly and concisely written with the effective use of underlines, and a revision is to be published every two years. This first edition seems to be fairly current on most subjects and offers a broad coverage, including material on bacterial cytology and physiology, variation, culture technics, chemotherapeutic and other antibacterial agents, as well as specific information on the most important pathogenic bacteria, rickettsiae, viruses and fungi.

In an effort to limit the size and therefore the cost of the book, the writers have been brief to the point of omitting some fairly important matters, i.e., there is no clear discussion of bacterial hemolysis on blood agar plates and in the section on blood groups no mention is made of the Rh factor. The discussion of the *Neisseria* group is extremely short, no more space being devoted to this than to the relatively unimportant pleuropneumonia-like organisms.

The order of chapters is faulty in one instance: allergy, hypersensitivity, and active and passive immunity are discussed before antigens and antibodies.

A major limitation of this text is the absence of specific references other than a list of general reference at the end.

However, the book is readable, concise, up-to-date, and seems to be especially good in its presentation of the material on viruses and rickettsiae.

WILLIAM A. PIERCE, JR., M. D.

Textbook of Pediatrics; by Waldo E. Nelson, M. D. (ed.) 6th ed., Philadelphia, Pa., W. B. Saunders Company, 1954, Pp. 1581, Price $15.00.

This edition is a continuation of the Griffith, Griffith-Mitchell series of text-books on Pediatrics. The foundation of the series was laid many years ago by Dr. Griffith of Philadelphia and the editorial responsibility was soon shared by Graeme Mitchell late of Cincinnati and at his death Waldo E. Nelson, who also joined Graeme Mitchell after the death of Dr. Griffith, assumed full responsibility for subsequent editions. The series for many years was confined to one volume, later it appeared in two volumes and is now presented in a single volume of 1581 pages.

As in most text-books of this magnitude Dr. Nelson has associated with him 72 contributing authors, each of whom are outstanding authorities in the special field of pediatrics on which they have written sections or chapters. The format has been very much improved over previous editions. The two-column page is used in keeping with what has now become a conventional style. It has long since been agreed that this style enables the reader to use a minimum amount of ocular effort because of the short lines.

The plan of the entire book as shown in the table of contents enables the reader quickly to obtain the information sought with the expenditure of a minimum amount of time and effort. The

text throughout the book is adequately illustrated with good photographs and a few colored plates.

This edition is dedicated to "The Doctor devoted to the welfare of children" as exemplified by Dr. Frank E. Stevenson, a colleague of the editor and contributors—whose influence is continued in the many physicians who as students, interns, and resident physicians saw in him an ideal.

This book will continue to serve students, general practitioners, and specialists in pediatrics just as previous editions have down through the years.

ROBERT A. STRONG, M. D.

Pediatric Diagnosis; by Morris Green and Julius B. Richmond, Philadelphia and London, W. B. Saunders Company, 1954. Pp. 436—xvii. Price $10.00.

In *Pediatric Diagnosis* the authors clearly summarize the diagnostic approaches used by the well grounded and thoughtful pediatrician. Skilfully woven into the text are certain therapeutic measures which the reader will find valuable additions to his armamentarium. The importance of growth and development as it effects the evaluation of signs and symptoms is clearly presented.

This book should appeal to a broad range of readers. The pediatrician will enjoy and profit by the excellent review. General practitioners will find reading this book an extremely painless way to improve his appraisal of both the sick and the healthy child. The medical student likewise will find this text most helpful when he is introduced to the complex phenomena presented by his first pediatric patients.

One of the virtues of the book is the fact that many controversial and unimportant details are omitted. The references are incorporated in the text and have been carefully selected. This innovation will serve to stimulate the reader's interest in the available current literature.

In general this is an excellent book. The authors are to be commended on their concise presentation of a broad and difficult subject.

JOHN H. DENT, M. D.

PUBLICATIONS RECEIVED

The Philosophical Library Inc., N. Y.: Pomp and Pestilence; Infectious Disease, Its Origin and Conquest, by Ronald Hare, M. D.; Management of Addictions, by Edward Podolsky, M. D.

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